AEROBIC EXERCISE AND SLEEP

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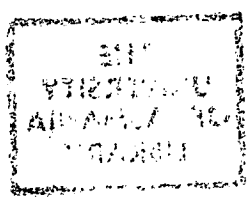

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ABSTRACT

The restorative and energy conservation theories of sleep predict that physical exercise will result in increased slow wave sleep (SWS) and sleep duration. Restorative theories hypothesize that exercise creates a need for restoration, with a concommitant increase in SWS and sleep duration; while the energy conservation hypothesis holds that sleep, and SWS in particular, is a state of energy conservation, such that, any increase in energy expenditure caused by exercise should result in an increase in these components of sleep. Exercise may be acute (a particular exercise session), or chronic (physical fitness due to habitual exercise). Accordingly the effects of exercise on sleep may be a consequence of a particular exercise session, or physical fitness.

The present literature does not show clear support for a facilitative effect of exercise on SWS or sleep duration. There is evidence however, that the effect of exercise may depend on the fitness and age of the subjects and the intensity and duration of the exercise. This thesis reports three experiments designed to assess the hypothesised facilitative effect of exercise on SWS and sleep duration in young and older fit subjects under varying conditions of intensity and duration of exercise.

Physical fitness resulting from aerobic training, has been shown to be associated with higher levels of SWS and longer sleep duration in young subjects. It was of interest to determine if this finding extended to an older population. The effect of aerobic training in young and older subjects was compared in the second experiment.

In the first experiment the sleep of 11 fit young subjects was compared across 4 conditions: a no exercise condition, a one hour walk, a one hour run, and a 6 hour walk. In the second, the sleep of four groups of subjects, 10 younger fit and 10 younger unfit subjects (average age 22 years), and 12 older fit and 9 older unfit subjects (average age 41 years) was compared under no exercise conditions, and the sleep of the fit subjects was compared under a no exercise and a severe ($1\frac{1}{2}$ hour training run) exercise condition. Finally, in the third experiment, the sleep of 8 marathon runners (average age 40.75 years) was studied following a no exercise condition, a $1\frac{1}{2}$ hour training run and a competitive marathon.

The results indicated that exercise did not increase SWS or sleep duration in any condition in any experiment. Instead, the second and third experiments indicated that intense exercise disrupts sleep. In the second experiment aerobic fitness in both age groups was associated with increased SWS, decreased sleep onset latency, and a tendency to increased sleep duration. This latter result replicates and extends previous findings of the effects of fitness on sleep.

The negative results clearly fail to support either body restorative or energy conservation theory in their present form. The validity of the observation that aerobic fitness affects sleep is extended since it has been found in both young and older subjects. However, this finding does not offer support for either theory as other studies have recently shown that the fitness effect is limited to aerobic training.

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TABLE OF CONTENTS

LIST OF TABLES

LIST OF FIGURES

CHAPTER 1

INTRODUCTION

CHAPTER I

INTRODUCTION

Sleep is a mystery and a wonder to most of us, even, or especially, to sleep researchers, and as we understand more about sleep the experience of sleep will remain mysterious for many. As a scientific problem sleep will provide us with different puzzles. It represents the culmination of many processes and as a consequence there are many theories about sleep, and about the functions of sleep. Such theories range in level from the fine grained biochemical and neuroanatomical to those concerned with the psychological effects and the physiological characteristics of sleep. In this latter case sleep characteristics such as amount of sleep are often thought to be related to the organisms' adjustment to its environment. This thesis is a study in the latter area. It concerns the relationship between aerobic exercise and sleep, and addresses the immediate effect of an exercise session on sleep, and the effect of fitness on sleep.

Exploration of the relationship between exercise and sleep has for sleep research a relatively long history which originated in theories that argue that non rapid eye movement (NREM) sleep is related to restorative processes and energy conservation (Oswald, 1962; Fisher, 1965; Roffwarg, Muzio and Dement, 1966; Zepelin and Rechtschaffen, 1974; Berger, 1975). The first empirical study in the area was that of Baekeland and Lasky (1966) and they found an increase in slow wave sleep following an afternoon's exercise session. This finding is what is known as 'the exercise effect'. Baekeland and Lasky (1966) also suggested the possibility of a difference in sleep patterns or sleep

architecture between fit and unfit individuals, an effect subsequently confirmed by Griffin and Trinder (1978). The Baekeland and Lasky (1966) study was the stimulus for work examining the relationships between exercise and sleep. Experimental studies of the effects of exercise on sleep are reviewed in chapter 2. The empirical complexities of the relationships between exercise and sleep are explored, and the empirical rationale for the experiments reported in subsequent chapters are introduced.

In considering the effects of exercise on sleep there is a need to take into account a variety of complex processes. These include the physiological effects of exercise and how these may effect sleep, and the effects of age on sleep. Our understanding of these processes and their relevance in the context of the thesis is described in chapters 4 and 5: Chapter 4 discusses relevant exercise physiology including the effects of exercise, the effects of training, and the effects of exercise and training with age. Chapter 5 is concerned with the changes in sleep that occur with age. This is particularly relevant because of the age range of the subjects used in the experiments reported in chapters 6, 7 and 8.

Recent studies of sleep have posited a number of theoretical claims which have been heuristic. Among these have been the various restorative views, and that of energy conservation (Zepelin & Rechtschaffen, 1974; Berger 1975). There are in essence two forms of restorative theory of sleep: the bodily restorative view (Hartman, 1973; Oswald, 1974; Adam and Oswald, 1977), and the central nervous system restorative view (Feinberg, 1974, Horne, 1979, 1981, 1983). The first

holds that sleep is a necessary state for the restoration of bodily functioning including the central nervous system, and the second that sleep is necessary for the restorative processes of the central nervous system but not for restorative processes of bodily functioning. One approach to the study of both restorative and energy conservation theories has been to study any changes in sleep following exercise. The ideas that sleep allows bodily restorative processes to take place, and that sleep might also fulfil energy conservation roles are central to the development of this thesis, and it is work based on these notions that is reported here. The theoretical views of restoration and energy conservation and their relevance to sleep and exercise are discussed in chapter 3. The issues concerned with the demonstration of an exercise effect in subjects from two different age groups, and the theoretical and empirical implications of the studies reported here are considered in the final chapter.

CHAPTER 2

THE EXERCISE EFFECT

CHAPTER 2

THE EFFECT OF EXERCISE AND PHYSICAL FITNESS ON SLEEP

Restorative theories of sleep and the notion that exercise has a facilitative effect on sleep especially on slow wave sleep (SWS) have an intertwined history such that the search to define a facilitative effect on sleep has been an important element in the search for support for bodily restorative theories of sleep. Originally at least, bodily restorative theory predicted that exercise increases the amount of SWS and perhaps the total amount of sleep. Baekeland and Lasky (1966) showed such a SWS effect in fit subjects, and suggested the differentiation between the effects of an exercise session (an exercise effect) and the effects of fitness (a training or regular exercise effect) on sleep. In the first section of the chapter it is proposed to consider the empirical support for a facilitative effect of exercise on SWS and sleep duration, while in the second section the effects of physical fitness will be evaluated.

The Exercise Effect

Including the Baekeland and Lasky study there have been about 25 experiments which have considered the effects of exercise on sleep. The exact number varies depending on whether those which use exercise in conjunction with sleep deprivation (e.g. Moses, Lubin, Naitoh and Johnson, 1977) as the independent variable are taken into account. Here I am concerned to elucidate the effects of exercise on sleep in which the dependent variable is a part of the normal daily cycle.

Of the experiments that have considered the effects of exercise on sleep relatively few have managed to show an effect of exercise on SWS or the amount of sleep. Of the experiments reported in Tables 2.1, 2.2 and 2.3, only about 25% of the studies have found clear cut effects, although some others have shown ambiguous, or small effects. It is possible that these findings may be a consequence of the exercise effect being dependent on other factors such as:

fitness,

age,

sex,

and exercise variables such as:

the intensity and duration of exercise,

and time of day of exercise.

Fitness

Of the variables mentioned above one that captured initial interest as an explanation for conflicting findings was the fitness of the subjects. Griffin and Trinder (1978) confirmed the speculation of Baekeland and Lasky (1966) demonstrating that fit subjects after an afternoon's exercise session had an increase in stage 3 sleep (although not a significant increase in total SWS) and a decrease in the latency to SWS, while unfit subjects did not show such an effect.

In studies that enable consideration of the effects of exercise on unfit subjects, it is clear that unfit subjects do not usually show facilitatory effects of exercise on sleep. Of twelve studies (Tables 2.1

and 2.2) which used unfit subjects, four demonstrated such effects: one (Bunnell, Bevier & Horvath, 1983) demonstrated an increase in SWS, Browman (1980) showed an increase in 1st cycle SWS and a decrease in sleep onset latency (SOL), Horne and Porter (1975) showed an increase in stage 3 of sleep in the 1st cycle and one showed an increase in total sleep time (TST) (Zir, Smith & Parker, 1971). The remaining eight studies showed either no exercise effects or detrimental effects.

In sum, unfit subjects do not normally show an exercise effect. Where unfit subjects evinced facilitatory effects of exercise on sleep this might be a function of variables such as the type of exercise used, or sex of the subjects. The effect of these variables will be considered later.

Studies of fit subjects have more frequently shown an exercise effect (see Table 2.2 and Table 2.3). Some have demonstrated facilitative effects of exercise on SWS and sleep duration (Baekeland and Lasky, 1966; Shapiro, Griesel, Bartel and Jooste, 1975; Maloletnev, Telia and Tchatchanashvili, 1977: Griffin and Trinder, 1978; Shapiro, Bortz, Bartel and Jooste, 1981) whereas others have not (Baekeland, 1970; Walker, Floyd, Fein, Cavness, Lualhati and Feinberg, 1978; Bonnet, 1980; Trinder, Bruck, Paxton, Montgomery and Bowling, 1982; Paxton, Trinder and Montgomery, 1983), although Baekeland (1970) showed a tendency to increased SWS. One of the studies showing a SWS effect reported that this was primarily an increase in stage 3 of sleep (Griffin and Trinder, 1978).

Table 2.1

Studies using Unfit Subjects
in Single Group Designs

Authors	Subjects	Level of Exercise	Sleep Effects
Adamson et al.(1974)	12M 5 fit 7 unfit* 19-31 yrs	more than usual	no effect
Browman and Tepas (1976)	9M 19 yrs	light dynamic 20 km. on bicycle erg.	SOL inc. REM Lat inc.
Browman (1980)	4M 3F 19-22 yrs	Static at 40% VO2 max 2 X 40 min sessions	SOL dec 1 st cycle SWS inc
Bunnell (1983)	4M 5F	dynamic to volitional exhaustion	SWS inc.
Desjardins et al.(1974)	6M	high intensity and low intensity exercise	REM % dec both conditions
Hauri (1968)	15M	6 hrs strenuous	no effects
Horne and Porter (1975)	8M	45% VO2 max for 85 min on bicycle erg.	stage 3 inc in 1st cycle
Paxton et al.(1982) (Expt.1)	14M 10F 4 groups	light to medium hard varied across groups	no sleep effects
Zir et al. (1971)	10M	light exercise in 5 medium exercise in 5	inc in mean TST

* subjects treated as a single group.

Abbreviations: dec = decrease; erg = ergonometer; F = female subjects; hrs = hours; inc = increase; lat = latency ; M = male subjects; min = minutes; yrs = years. These abbreviations are used in other tables. Sleep abbreviations are found in the text.

Table 2.2

Studies that compare fit and unfit subjects

Authors	Subjects	Level of Exercise	SWS Effects
Griffin and Trinder (1978)	8fit 4M 4F 8 unfit 4M 4F	7.3 km. difficult run	Fitness effect Stage 3 Fitness by exercise effect. No exercise effect in unfit subjects
Paxton et al. (1983)	8M fit 9M unfit	fit- exhausting but stressful. Unfit- 4-6 km.	no exercise effects
Walker et al.(1978)	10M fit 10M unfit	10.2 km. run 2.4 km. run	no exercise effect in unfit or fit

One factor which makes the problem of interpretation of studies using fit or unfit subjects more difficult is that disturbed sleep frequently follows exercise (cf. Baekeland and Lasky, 1966). This effect is shown by increased sleep onset latency (SOL) (Browman and Tepas, 1976), decreased rapid eye movement (REM) (Desjardins, Healey and Broughton, 1974; Shapiro et al., 1975; Bonnet, 1980; Shapiro et al., 1981), delayed REM latency (Browman and Tepas, 1976) and increased wake time (Shapiro et al, 1981). Such disturbances in sleep may be due to physiological arousal, blisters, muscle and joint soreness and discomfort induced by metabolic changes and insufficiencies including dehydration following considerable physical effort. Conditions producing disturbed sleep may also mask potential increases in SWS and sleep duration. If so this would account for the greater success rate with fit subjects as they would be less likely to be stressed by exercise.

The data of Buguet, Roussell, Angus, Sabiston and Radomski, (1980) whose results related sleep architecture to adrenocortical steroid secretion are consistent with this interpretation. They showed that an increase in the secretion of these hormones would decrease stage 3 of sleep and that fit and healthy male subjects could be differentially affected by prolonged sub-maximal exercise. In four of the Buguet et al. (1980) subjects there was a stage 3 increase as an effect of exercise with no change in steroid secretion and in the other two subjects an increase in steroid secretion with no increase in stage 3 of sleep. In the latter two subjects the increase in steroid secretion may indicate an increase in arousal or a stress effect of the exercise.

In summary, of all the studies thus far discussed there is no convincing evidence for an exercise effect in unfit subjects. However, in

fit subjects about half the reported studies managed to show such a SWS effect. The reason for the conflicting results may lie in the stress effects of the exercise, or it may be due to the confounding of exercise with some other variable or variables.

Age

In all of the studies thus far reported only one (Trinder et al., 1982) used older subjects. This study failed to show an exercise effect and one possible explanation for this conflicting result is that older subjects may have their sleep disrupted by exercise. Trinder et al. (1982) compared the sleep of fit and unfit subjects from two age groups, one younger (mean age 22.0), and one older (mean age 31.8), following conditions of exercise and non exercise. Their data suggests that while the fit subjects spent more time in bed and had a longer sleep period time, the older fit subjects spent more of this extra time either awake, or in relatively disturbed sleep. The findings imply a limitation on the age at which exercise may effect sleep, and in view of the implications including theoretical, experimental and medical, replication of the study seems most desirable.

Sex

In the total sleep and exercise literature there are only four studies which have used female subjects (Browman, 1980; Griffin and Trinder 1978; Trinder et al., 1982; Bunnell et al., 1983). Two of these studies report that women show greater facilitative effects of exercise on sleep (SOL, Browman, 1980; and SWS, Bunnell et al., 1983). In addition

Table 2.3

Studies of Fit Subjects

Authors	Subjects	Exercise	Effects on sleep
Baekeland (1970)	14 males	Usual	Tendency to inc SWS
Baekeland and Lasky (1966)	10 males	Usual	inc. in SWS
Bonnet (1980)	12	march 6.5hr	dec REM
Buguet et al. (1980)	6	6 x 34 km/d	individual differences
Shapiro et al. (1975)	2	graded to intense	inc SWS dec REM
Maloletnev et al. (1977)	15	intense	inc stage 4
Shapiro et al. (1981)	6	intense/long	TST inc SWS inc REM SOL WAKE dec
Trinder et al. (1982)	6 young fit 6 young unfit 6 older fit 6 older unfit	fit only usual	no sleep effects

Trinder has recently reanalysed the data from Griffin and Trinder (1978) (Trinder, personal communication) and this indicates that women show increased SWS following exercise compared to men. However in Trinder et al. (1982) there was only one woman in each group of 6 subjects, and separate data was not reported for these subjects.

As noted by Bunnell et al. (1983) the results of studies on female subjects need to be pursued, since the findings from three out of four experiments suggest that female subjects may be more susceptible to facilitative effects of exercise on sleep. Further, in future experiments using both men and women subjects the data should be analysed separately for men and women in order to avoid possible confounding.

Intensity and Duration of Exercise

The total amount of exercise used in a particular experiment can be viewed as a product of the duration of the exercise and the intensity or rate of energy expenditure. These two factors have not been varied independently, though, on the basis of across study comparisons, Horne (1981) has suggested that the rate of energy expenditure might be the more critical factor in producing a facilitative effect of exercise on SWS.

In most of the studies in the exercise literature on fit subjects it is possible to assess within reasonable limits the intensity and duration of the exercise. There are four studies in which intense exercise with high rates of energy expenditure have been used (Desjardins et al., 1974; Shapiro et al., 1975; Maloletnev et al., 1977; Shapiro et al., 1981) and three of these have produced facilitative effects of exercise on sleep

(Shapiro et al., 1975; Maloletnev et al., 1977; Shapiro et al., 1981). No facilitative effects were reported by Desjardins et al. (1974), but the study is more difficult to evaluate since duration and intensity of the exercise are not well described. The most direct and substantial evidence supporting the proposal derives from Shapiro et al. (1981). This study used a sample of six young distance runners, with a mean age of 21.7 years, who completed a 92 km marathon. The results show a marked increase in SWS and an increase in TST following the run.

Three studies have considered exercise of low intensity and long duration (Desjardins et al., 1974; Bonnet, 1980; Buguet, et al., 1980). There were no overall facilitative exercise effects on SWS or sleep duration in the studies, although Bonnet reported a 1st cycle SWS increase and there was significant REM reduction following exercise suggesting some sleep disturbance.

The available literature on intensity and duration of exercise suggests that duration of exercise of itself is not a factor likely to facilitate SWS and sleep duration, but that exercise of high intensity over a sufficiently long duration produces a facilitative effect on sleep. It is important to disentangle the two variables and to assess what duration of high intensity exercise will produce the effect. However it is also important to consider the stress of high intensity exercise since it is clear that such stress can disrupt sleep.

Time of Exercise

The time of day of the exercise is also potentially important in establishing an increase in SWS and sleep duration following exercise, since too great a lapse as in morning exercise may mean recovery takes place before sleep, and high arousal resulting from evening exercise may disrupt sleep. Two studies have used evening exercise only (Hauri, 1968; Desjardins et al., 1974) and each of these failed to show an exercise facilitation effect on sleep. Only two studies have varied time of day of the exercise (Baekeland and Lasky, 1966; Horne and Porter, 1975). Horne and Porter used subjects of average fitness under conditions of morning exercise and afternoon exercise. They managed to show no overall effect of time of day of exercise on sleep although they did establish an increase in stage 3 of sleep following afternoon exercise in the first half of the night. Only Baekeland and Lasky (1966) varied time of day in fit subjects, and in their study SWS was greater after afternoon exercise than evening exercise.

As far as can be assessed most other studies have used afternoon exercise. In the light of the potential confounding of an exercise effect by the time at which the exercise takes place it appears sensible to use only afternoon exercise unless studying time of exercise.

First Cycle Effects

During the course of the previous discussion the results of some studies have indicated that exercise may effect sleep primarily in the 1st

part of the night. This has lead to analyses of the 1st sleep cycle, or the 1st third or half of the night. The studies which have done such analyses are shown in Table 2.4

All of the four studies which have shown a facilitative effect of exercise on SWS early in the night used young subjects. Thus there was an increase in SWS (Browman, 1980; Bunnell et al., 1983; Maloletnev et al., 1977) and stage 3 of sleep (Horne and Porter, 1975). In two of the studies female subjects were used as part of the sample (Browman, 1980; Bunnell et al., 1983). Further, two of the studies used intense exercise (Bunnell et al., 1983; Maloletnev et al.,1977).

In the remaining six studies, one indicated disruptive effects of exercise on REM sleep (Desjardin et al., 1974), and one showed SOL increases (Browman and Tepas, 1976). The other four studies showed no effects of exercise on early sleep. The possibility of a facilitative effect of exercise on early sleep is thus inconclusive but again is possibly masked by other factors such as the intensity of exercise and sex of the subjects.

Taking the exercise and sleep literature as a whole a facilitative effect of exercise on SWS and sleep duration has only been observed regularly in young fit subjects. However, even with these subjects it is still not clear under what conditions such an effect takes place, and only one study has been conducted with older subjects. Thus factors which may influence the exercise effect and which require clarification include the age of the subjects, sex of the subjects, and intensity, duration and time of day of the exercise.

Table 2.4

1st Cycle Effects or
Early Hours of Sleep Effects

Authors	Cycle or Hours	Effects
Browman (1980)	1st cycle	SWS inc MT dec
Browman and Tepas (1976)	1st 2.5 hrs	SOL inc.
Bunnell et al.(1983)	1st cycle	SWS inc REM dec
Desjardins et al.(1974)	1st 3 hrs	REM% dec
Hauri (1968)	1st cycle	No effects
Horne and Porter (1975)	1st cycle	Stage 3 inc in morning exercise 2 inc
Maloletnev et al.(1977)	1st cycle	SWS inc
Paxton et al.(1982)	1st cycle	No effects
Paxton et al.(1983)	1st cycle	No effects
Zir et al.(1971)	1st 3 hrs	No effects

The Fitness Effect

As noted previously Baekeland and Lasky (1966) suggested a differentiation between the effects of exercise and the effects of fitness on sleep. This section considers effects of fitness on sleep.

There have been only a few experiments which consider the possibility that physical fitness may result in higher levels of SWS or longer sleep duration by directly comparing the sleep of fit and unfit subjects. Some of these studies have found more SWS in young aerobically fit athletes (Griffin and Trinder, 1978; Trinder et al., 1982; Trinder, Paxton, Montgomery and Fraser, 1985) when compared to sedentary controls although two have not (Walker et al., 1978; Paxton, Trinder, Shapiro, Adam, Oswald and Graf, 1984b).

It is possible that the failure of Walker et al. (1978) and Paxton et al. (1984b) to show higher levels of SWS in fit subjects relates to type of training, diet or other factors. Recent work indicates that fitness is only one of a number of possible related factors in SWS differences. Thus body composition (Paxton, Tinder, Montgomery, Oswald, Adam and Shapiro, 1984a; Paxton, Trinder, Shapiro, Adam, Oswald and Graf, 1984b), type of training (Trinder et al., 1985) and diet and life style factors (Paxton, Trinder and Montgomery, 1983) may effect SWS.

While Walker et al. (1978) failed to show a SWS difference between their fit and unfit groups, they did show more TST in their fit as opposed to the unfit subjects in an exercise condition ($.05>p>.01$). In addition, in a reanalysis of five experiments (one of which is Experiment 1 in Chapter 6). Montgomery, Trinder and Paxton (1982)

found that SWS and TST are greater in physically fit subjects. Thus it appears that physical fitness may be related to both SWS and TST but the exact nature of the relationship is not clear.

It is noteworthy that Trinder et al. (1982) failed to show the fitness effect on either SWS or TST in older subjects (mean age 31.8 years). As all other experiments considering the phenomena used young subjects this suggests that the fitness effect might be restricted to the younger age group.

In general terms it appears that physical fitness is related to SWS and to sleep duration. One possible factor influencing these finding is that of age and thus replication of Trinder et al. (1982) seems desirable.

Conclusion

A number of variables have been identified which may affect the facilitative effect of exercise on SWS and sleep duration. The studies to be reported in this thesis were concerned with the effects of the intensity and duration of the exercise and the age of the subjects. In addition, the physical exercise effect in young and older subjects was compared. Later chapters report the studies involved. Before presenting these studies other background material is considered, with the next chapter describing theoretical views relevant to the effects of exercise on sleep.

CHAPTER 3

THEORIES OF SLEEP

CHAPTER 3

THEORIES OF SLEEP: RESTORATION AND ENERGY CONSERVATION

The exercise studies reported in the previous chapter have been conducted because of their relevance to theories of sleep. In particular exercise has been seen as a way of evaluating two general theoretical positions: that sleep might serve the roles of restoration and/or energy conservation. The notions that sleep is restorative and that it may serve an energy conservation role are of course not mutually exclusive, indeed Baekeland and Lasky (1966) hypothesize that SWS sleep is related both to energy expenditure and to physiological restoration. Both these theoretical propositions are discussed in this chapter.

Restorative Theories

The idea that sleep is restorative appears embedded in our culture and many books and theses on sleep use an apt quote showing at least the general view among many great writers that sleep is restorative. It is only recently that such theories of sleep have been scientifically assessed, and that they have been developed to the level of sophistication that allows comparison between the theories in the laboratory.

There are two main forms of restorative theory, general or bodily restorative theories (Hartmann, 1973; Adam and Oswald, 1977, 1983; Oswald, 1969, 1974, 1980) which propose that sleep is restorative for both body and brain, and brain or central nervous system restorative theories (Feinberg, 1974; Horne, 1977, 1979, 1980, 1981, 1983).

Bodily Restorative Theories

The main proponents of this view are Hartmann (1973) and Adam and Oswald (Adam, 1980; Adam and Oswald, 1977, 1983; Oswald, 1969; Oswald, 1974; Oswald, 1980), although it is Adam and Oswald who have contributed most to this view in recent years.

Hartmann (1973) posits that there are two sleep requirements, one for SWS and one for REM sleep, and that each of these two types of sleep performs a different but perhaps related function. He suggests that there is a relatively unchanging need for SWS and that at least one function served by the processes occuring within this sleep form is that of anabolism especially macromolecular synthesis. In part it is proposed that the need for restoration is increased after exercise, injury and physical tiredness and consequently SWS increases. On the other hand, Hartmann (1973) hypothesizes that during REM sleep synthetic products from SWS are used in repair and restoration of synaptic connections and also in the formation of new connections in the catecholaminergic neuronal systems required for focussed attention and learning. Accordingly REM has a CNS restorative role. In general terms the notion of dual functions of sleep proposed by Hartmann is similar to that proposed by Oswald initially in 1969.

Oswald (1969) suggested that sleep is a time for restoration of the body and brain. Sleep is seen as an unresponsive state during which the whole body including the nervous system can recuperate (Adam and Oswald, 1977). Work by Adam and Oswald has elaborated this view and

they have postulated processes by which such restoration might take place. In general it has been argued that SWS has the chief function of general bodily restitution and that REM sleep is chiefly for brain restitution (Oswald, 1969,1976). Such restitution or repair depends upon protein synthesis. Adam (1980) and Adam and Oswald (1977) postulate that the lowered external energy demands on the organism during sleep allow the build up of energy within cells and that high cell energy levels stimulate protein synthesis. Adam and Oswald (1977) argue that the rest/activity cycles in simple organisms and sleep/wake cycles in higher animals each reflect cycles in available cell energy. During wake there is a high demand for energy because of the more active behaviour. This energy demand is met by degradation of available energy sources and relatively little in the way of restoration can take place. However during sleep there is less active behaviour, a lowered energy demand on cell processes and accordingly high energy levels available to stimulate restoration and protein synthesis (Adam and Oswald,1977).

There are three factors which determine the rate of protein synthesis,

i) the availability of substrate materials through food and body stores,

ii) the availability of energy for the synthetic processes, and

iii) the concentration of synthetic end products (Adam, 1980).

Of these three factors it is probably the availability of energy which is most important in their hypothesizing.

A cell's activity is controlled via its available chemical energy which is stored in the form of adenosine triphosphate (ATP) with adenosine diphosphate (ADP) and adenosine monophosphate (AMP) being

metabolic products of the release of energy from ATP. In somewhat crude terms a cell's chemical activities can be divided into those that use energy and thus deplete ATP and those that produce ATP and thus store energy. The energy charge (EC) of a cell can be measured by the relative amounts of each of ATP, ADP and AMP in the cell. The balance of the energy charge within a cell has been defined by Atkinson (1968) as EC = (ATP + ADP/2) / ATP + ADP + AMP.

The EC varies up to unity but has an operative physiological range of between 0.70 and 0.95 (Atkinson, 1970). The operation of cell enzymes under conditions of low EC values favour the production of ATP from nutritive materials and thus degradation of proteins, and when work decreases, degradation decreases and EC and ATP increase. During this later phase protein synthesis is stimulated since protein synthesis requires a high level of EC. Adam and Oswald (1983) argue that it is not that synthesis requires a lot of energy but rather that it requires a high level of EC. Thus as EC varies so does the operation of control enzymes in synthetic and degradative pathways as described in the following two diagrams (Figures 3.1 & 3.2; Adam, 1980).

It should be noted from the diagrams that rapid changes in the rates of synthesis and degradation can occur with small changes in EC, and of course, the two processes are linked in a reciprocal fashion such that the sum of the processes will show a rhythm of activity.

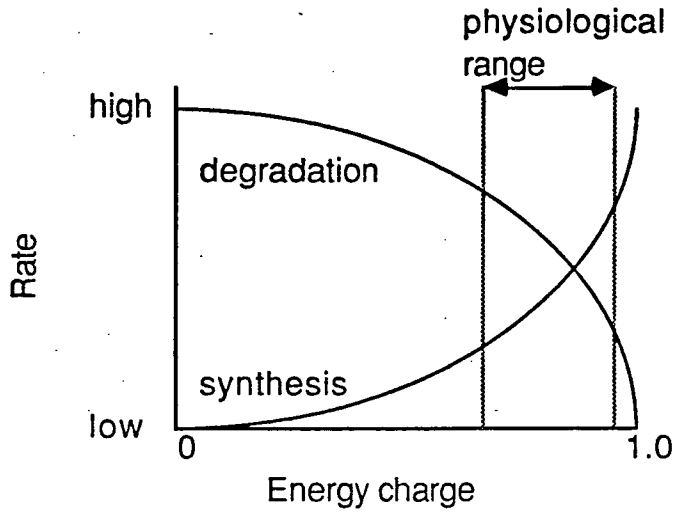

Figure 3.1. "Typical curves of the rates of reaction of control enzymes in synthetic and degradative pathways in response to different levels of cellular energy charge." (Adam, 1980, p.291)

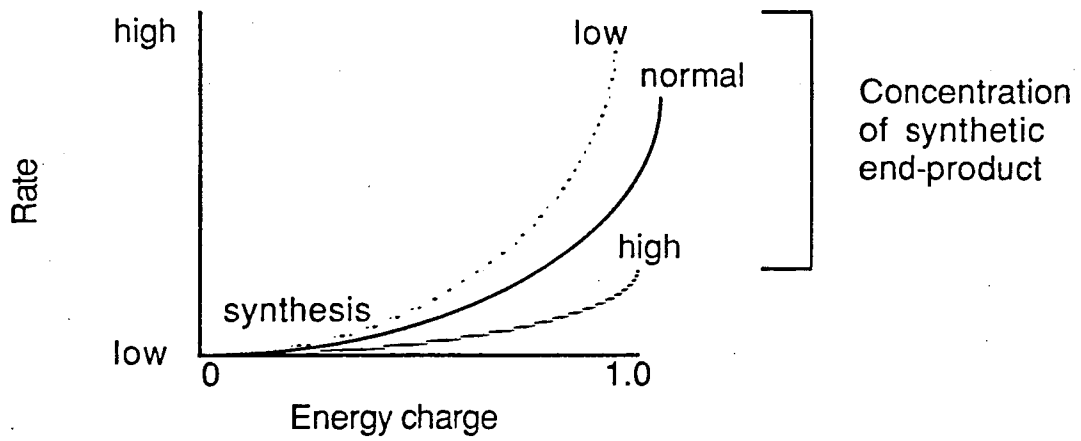

Figure 3.2. "Typical rate of reaction of a synthetic pathway in response to different levels of cellular energy charge and the modification of this response by high and low concentration of synthetic end-product." (Adam, 1980, p.291)

While the basis of these notions is derived from the behaviour of cells it is argued (Adam,1980) that the same principles apply in complex organisms. Accordingly, during SWS metabolic rate is at its lowest level and thus EC will be high creating the situation where there will be rapid rates of protein synthesis, anabolic repair and mitosis.

In discussion of the bodily restorative theories there is the explicit notion that restoration (and degradation) via protein synthesis takes place all the time, but that the relative proportions of degradation to restoration change such that sleep is the time of most restoration and compensates for the intensity of waking activity and that slow wave sleep is of more restorative value than stages 1 and 2 (Adam and Oswald, 1977; Adam, 1980; Oswald, 1980). Then clearly more time asleep will lead to greater restoration as will a shorter time of less responsive (or more intense) sleep. Adam (1980) postulates that during SWS there are enhanced changes in the rate of synthesis. It follows from this that an increase in SWS, or an increase in the intensity of sleep, but not necessarily in the total length of sleep will be related to greater restoration. Thus both the total sleep time and SWS need to be taken into account in restorative processes.

Adam and Oswald's restorative model predicts that any process which creates extreme depletion of energy charge would be followed by a corresponding period of considerable lowering of metabolic rate with a concommitant increase in cell energy charge, in anabolic processes and in SWS and sleep duration.

The evidence in support of the importance of SWS as a period of restoration has been documented by Adam and Oswald (1977), Adam

(1980) and Oswald (1980). A summary of the major points of this evidence follows.

i) Situations of altered metabolic demands such as exercise and some abnormal physiological conditions show an increase in TST and or SWS.

   a) After strenuous exercise plasma growth hormone (GH) (Adamson, Hunter, Ogunremi, Oswald & Percy-Robb, 1974) and SWS (see Chapter 2 for review) have both been found to increase.

   b) During periods of fasting there is an increase in SWS and in the secretion of plasma human growth hormone (Parker et al, 1972) which is associated with anabolic repair. There is also an increase in SWS in anorexic patients undergoing refeeding and who therefore are in an anabolic state (Lacey, Crisp, Kalucy, Hartman and Chen, 1975).

   c) Hypothyroid patients have markedly reduced SWS compared with normal controls (Kales, Heuser, Jacobson, Kales, Hanley, Zweizig, and Paulson, 1967) while hyperthyroid patients show excesses of SWS (Dunleavy, Oswald, Brown, and Strong, 1974). Thyroid hormone increases degradation (Adam and Oswald, 1977) and when hyper- and hypo- thyroid patients undergo corrective therapy SWS essentially returns to normal (Dunleavy, et al., 1974; Kales, et al., 1967).

ii) There is a correlation between growth hormone secretion and SWS (Takahashi, Kipnis and Daughaday, 1968; Sassin, Parker, Mace, Gotlin, Johnson and Rossman, 1969; Parker, Sassin, Mace, Gotlin

and Rossman, 1969) and over 70% of daily GH release occurs during the first two hours of sleep. In general, anabolic hormones (eg growth hormone, prolactin, luteinizing hormone and testosterone) are secreted in higher quantities during sleep suggesting that growth and restoration occur primarily during sleep (catabolic hormones such as the catecholamines and corticosteroids are secreted at the highest levels during active wake times).

iii) The process of cell mitosis for growth and tissue regeneration depends upon protein synthesis and the presence of higher cellular concentrations of ATP. This process occurs at a greater rate in some animal and human tissues during the normal sleep period.

The theory concerning SWS in the Adam and Oswald framework is relatively well developed and the theory predicts that increases in both sleep duration and SWS are both indicative of increased bodily restoration. In contrast, Oswald (1969, 1976) argued that REM sleep was indicative of a repair process which involved an increase in the intensity of brain synthetic activity. Since this work Oswald has paid less attention to the role of REM sleep and to its possible central nervous system restorative functions. However, other writers have continued to address the issue of brain restorative functions of REM sleep (eg. Drucker-Colin, 1979; McGinty and Drucker-Colin, 1982), but this set of problems do not bear on the relationship between sleep and exercise and will not be addressed here.

Central Nervous System (CNS) Restorative Theories

Feinberg's Central Nervous System Restorative Theory

Feinberg (1974) theorizes that sleep is a process that reverses the effects of waking activity in the central nervous system. His model is based on the following descriptive characteristics of human sleep which Feinberg views as indicative of the function of sleep.

i) Sleep is a cyclical phenomena such that SWS (including stage 2) invariably precedes REM sleep and that the two sleep forms occur in several discrete episodes instead of occuring separately and all at once.

ii) With increasing age the length of SWS becomes shorter, there is less stage 4 sleep, and the 1st REM sleep occurs earlier as SWS duration diminishes (Feinberg, 1974).

iii) The nightly decline in SWS sleep periods for children and young adults appears exponential, and the amount of SWS appears (within limits) dependent on the duration of preceding wakefulness, such that naps taken in the morning show little or no stage 4 sleep, while naps taken in the afternoon show stage 4. In addition, afternoon naps reduce the amount of stage 4 during the following night.

iv) Stage REM can be prolonged without decreasing REM the following night (Verdone, 1968; Karacan et al., 1970), and that REM seems to be able to be suppressed without obvious detriment to human subjects, thus he concludes that while 'REM appears dispensible, SWS seems essential' (Feinberg, 1974,p299).

Feinberg (1974) suggests that SWS and REM sleep have a functional relationship in which REM maximises the occurrence of SWS.

He hypothesizes two neuronal states in developing his model : State 1 (S1) a state present at the end of sleep and State 2 (S2) a state of depletion or degradation caused by waking. The first SWS period makes a substantial conversion from S2 to S1 but this process is not complete or optimal. REM however produces a substrate or cofactor substance which permits further SWS to occur and increase the level of S1. This process is repeated until optimal levels of S1 are achieved and is indicated by relatively flat EEG (stage 2), a lessened duration of SWS period and perhaps other factors such as the abrupt increase in eye movements after 7 1/2 - 10 hours sleep (Aserinsky, 1969).

The Feinberg (1974) model was speculative since it was based on descriptive characteristics of sleep and offers no information about the mechanisms involved or the particular physiological processes which were being restored. However it does offer an account of the cyclical nature of sleep and sleep cycle changes through life.

Horne's Restorative View

In a series of papers (Horne, 1977, 1979, 1980, 1981, 1983) Horne argues that sleep performs a brain restorative function and that it does not have a role in bodily restoration. In particular, his conclusion that sleep does not perform a bodily restorative role is based on an analysis of a number of research areas. This analysis includes the processes involved in sleep deprivation, sleep and growth hormone release, protein turnover during sleep and the concept of energy charge, and sleep and exercise. The following points are critical to his argument.

i) Horne argues that the EEG measures changes in brain function and does not measure changes in body tissue, particularly tissue

restitution (Horne, 1978,1981). Further he notes that the effects on sleep of experimental procedures such as exercise and sleep deprivation are primarily those assessed by the sleep EEG and by implication are brain function changes.

ii) The findings from sleep deprivation studies rather than indicating bodily deficits indicate a number of psychological deficits or symptoms such as behavioural irritability, suspiciousness, speech slurring, visual misperceptions of a usually minor kind which reflect CNS activities (Horne, 1978; Johnson, 1969).

iii) He believes growth hormone release in sleep is uncertain support for a tissue restoration hypothesis of sleep (Horne, 1983) He notes that growth hormone has a number of poorly understood functions including fat mobilization, the sparing of amino acids from gluconeogenisis as well as the promotion of protein synthesis. Further the purpose of growth hormone release in sleep is not known and may differ in function between children (who are still growing) and adults.

iv) In recent studies on human subjects (Garlick, Clugston, Swick and Waterloo, 1980; Clugston and Garlick, 1982) protein synthesis was found to be stimulated by food intake. In contrast during fasting as in sleep, protein synthesis was exceeded by protein breakdown, again suggesting that sleep is not a time of bodily restoration.

v) Horne (1983) argues that low metabolic rate in association with high cellular energy charge during sleep does not suggest high rates of restoration. He points out that protein synthesis is energy consuming. Thus if high levels of protein synthesis were occuring during sleep metabolic rate would not be low.

vi) Finally he argues that changes in sleep following exercise appear to occur only in fit subjects and that this poses a problem for the bodily restitution hypothesis since unfit subjects after exercise ought to require more restitution.

Horne (1983) concludes that the weight of evidence indicates that sleep is not a state of heightened restoration for body tissues with the possible exception of the brain. He proposes (Horne,1983) a two process view of sleep in which at sleep onset there are two sleep processes working in parallel, one of obligatory and restorative sleep oriented towards brain restitution, and a more facultative sleep drive perhaps permitting energy conservation, safety, and occupying the hours of darkness in which activity is difficult. The former would occupy only a few hours of sleep in the early hours of the night during which SWS usually occurs, and the latter would fulfill the process of maintaining sleep length for the time imposed by season, availability of food and safety (Horne, 1983).

In support of his views Horne (1977) has argued that with the evolution of the cerebrum and increasing body size the function of mammalian sleep may have shifted toward cortical restitution and concludes that if human sleep is oriented towards brain restitution then SWS could be associated with this restitution.

There is evidence that strongly suggests that SWS has an obligatory function. It is very difficult to alter SWS (Hartmann, 1973) and among environmental conditions only body heating, exercise, extended attention (Horne, 1985) and sleep deprivation seem to effect changes in SWS.

Further it is also very difficult to selectively deprive subjects of SWS - it tends to be maintained under partial sleep deprivation, it has priority in make up after total sleep loss and it is the sleep state which correlates best with length of prior wakefulness. On the other hand sleep can be both extended and reduced (Horne, 1983), suggesting a facultative component to sleep which enables man to adjust to seasonal changes and other environmental effects (Horne, 1983, 1985).

Although he is not specific about the nature of brain restorative processes, Horne (1979) suggests that both SWS and REM have equally important roles to play in cortical restoration and he notes that SWS in man appears to reflect an isolation and shutdown of cerebral function suggesting a recovery phase (Horne, 1983), although it may also be the effect on the brain of a deep torpor like sleep conserving energy (Horne, 1985).

Horne's original view argued that sleep including non REM and SWS serves a role of brain restitution and not that of bodily restitution (Horne, 1979). Indeed Horne and Porter (1976) concluded that exercise recovery processes began immediately after the exercise and intruded into sleep rather than requiring particular types of sleep. Thus the observation of an increase in SWS following exercise would have been considered as support for the bodily restorative view and incompatible with Horne's position. In 1981, however Horne noted the need to ask the question as to what effects the exercise might have on the brain and proposed the view that increased thermal load might increase SWS via an increase in brain temperature. The development of this view meant that both central and general restorative theories predicted increased in SWS

following exercise; one because the exercise increases the need for general bodily synthetic activity following the catabolism associated with the exercise and the other because the exercise increases brain temperature and stimulates metabolic activity. However it should be noted that Horne's view limits the conditions under which the effect will be observed to those which provide an adequate thermal load.

In summary Horne has suggested i) that both SWS and REM are important in cortical restitution but that SWS has an obligatory function; ii) that sleep might serve an energy conservation function. It should be noted that he has also indicated that sleep might serve to meet other demands such as safety.

Present versions of both the bodily restorative theory of Adam and Oswald and the brain restorative theory of Horne predict that SWS will increase following intense exercise and the bodily restorative theory also predicts an increase in TST under such conditions. In the next section it is proposed to discuss the energy conservation view of sleep which also makes predictions about the effects of exercise on sleep.

Energy Conservation

A central problem that confronts all homeothermic animals is that of maintenance of an energy balance (Berger, 1975). This problem is solved by various means and it is hypothesized (Berger,1975) that sleep is one of the processes that contributes to the solution by conserving energy, since during sleep the organism remains relatively immobile and unresponsive for a sustained period of time and thus has a reduced

demand for energy. The hypothesis that sleep contributes to energy conservation is widely recognized (Snyder, 1966; Berger, 1975; Berger, 1984; Walker and Berger, 1980; Zepelin and Rechtschaffen, 1974; Horne, 1977; Shapiro,1982). However the elaboration of this hypothesis has been largely due to the work of Berger (Berger, 1975; Berger, 1984; Walker and Berger, 1980).

It seems likely that the complete physiological and behavioural manifestations of sleep are unique to homeotherms (birds and mammals) and perhaps evolved in parallel with homeothermy and the consequent need for energy conservation (Berger, 1975). Berger (1975) argues that sleep represents one of a continuum of processes from inactivity to estivation and torpor through to hibernation. Thus sleep could be a variation of the other dormancy processes such as hibernation, daily torpor and estivation which are thought to have evolved as energy conservation processes (Berger, 1975).

Evidence for the energy conservation view comes from phylogenetic comparisons of sleep data, and ontogenetic sleep data. Berger(1975) notes that:

i) phylogenetic analysis shows that complete physiological and behavioural manifestations of sleep are unique to homeotherms and possibly that sleep evolved in parallel with homeothermy and the consequent need for energy conservation (Walker and Berger, 1980). Therefore sleep may be viewed as a period of dormancy analogous to hibernation, daily torpor and estivation (cf. Snyder, 1966; Allison and Van Twyver, 1970; Walker and Berger, 1980; Berger, 1984).

ii) an ontogenetic analysis shows both that SWS develops in parallel with the capacity of the newborn to maintain a relatively constant bodily temperature and that SWS is uniquely associated with homeothermy (Walker and Berger, 1980).

iii) general activity levels, heart rate (Kleitman, 1963;), body temperature and oxygen consumption (Kreider, Buskirk and Bass,1958) decrease during the course of the night in humans. In addition depth of sleep (excluding REM sleep) is inversely related to the rate of oxygen consumption (Brebbia and Altschuler, 1965). Zepelin and Rechtschaffen (1974) found a high positive correlation of 0.65 between total sleep time and metabolic rate with the implication that in animals with high metabolic rate there is a greater need for energy conservation. Accordingly such animals must have a high food intake or reduce metabolism by having inactive periods such as sleep, torpor or hibernation.

iv) sleep is highest in infancy (Williams, Karacan and Hursch, 1974) at the time of maximum growth so that it seems that sleep offers a method of energy conservation without retardation of growth (Berger,1975).

v) SWS increases in adults following starvation (Karacan, Rosenbloom, Londono, Salis, Thornby and Williams,1973; McFaydyen, Oswald and Lewis, 1973) and thus would seem to serve an energy conservation function (Berger, 1975).

vi) as brain and bodily metabolism decline with increasing age (Feinberg and Carlson, 1968) there is a parallel decrease in total sleep time and in the proportion of sleep spent in SWS (Williams, Karacan and Hursch, 1974; Blois, Feinberg, Gaillard, Kupfer and Webb, 1983).

Thus it is argued that SWS in particular has evolved as a period of reduced metabolism to partially offset increased energy metabolism while retaining a high enough body temperature to be able to respond to critical stimulation (Walker and Berger, 1980).

The development of this theoretical position has continued along several lines. In particular the work of Berger and Walker and their colleagues has elaborated the view that sleep is functionally related to torpor and hibernation, and hence to energy conservation (e.g. Walker and Berger, 1980). This has involved the analysis of the phylogenetic correlations between sleep and decreased metabolism and the correlation between sleep and metabolic rate within species (Berger and Walker,1980). Of particular interest are the correlations derived from available data on humans in which it is concluded that individuals with high metabolic rates sleep more than those with low rates (Walker and Berger, 1980). This is despite the prolonged periods of relaxed wakefulness enjoyed by man (Horne, 1977) and the small oxygen consumption savings (5 - 10 % drop in metabolic rate) in the sleep of man (Brebbia and Altschuler, 1965; Webb and Hiestand, 1975).

The energy conservation theory of sleep predicts that as both total sleep time and SWS are energy conservation processes, high levels of activity without compensatory increases in caloric intake, should result in their increase. However, the model is not precise with respect to the time span over which this adjustment will occur. Thus the relationship between activity level and sleep may be stated in at least three ways: that species differences in sleep are a function of species differences in achieving energy balance and these are in part a function of their

activity levels; that individual differences in sleep within a species are a function of long term energy balance and thus in part on long term activity levels; and that within species differences in sleep are a function of short term changes in activity levels. Eastman and Retchschaffen (1979) explored the latter two hypotheses. The report was an attempt to elucidate the effects of thyroxine on sleep in the rat and failed to find changes in total sleep time and activity as a result of the administration of thyroxine. They argued that while there may be interspecies differences in sleep as a function of energy demands, sleep does not appear responsive to either long term or short term variations of energy expenditure within individuals. In contrast Montgomery et al. (1982) reported an increase in sleep duration in subjects who were habitually exercising but not following short term variations in exercise patterns. These data are consistent with the view that sleep variables are slow to respond to changes in metabolic factors (Dunleavy et al., 1974).

Summary

Two general conclusions drawn from the theoretical positions presented in this chapter are as follows. First, bodily restorative and energy conservation theories predict that both sleep duration and SWS increase as a result of exercise. Second, Horne would predict no direct relationship between exercise and SWS or sleep duration. However, on the basis of his views on thermal load and his experimental work (Horne and Staff, 1983), he predicts that under conditions of increased core body temperature exercise will result in increased SWS.

CHAPTER 4

PHYSIOLOGY AND EXERCISE

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Exercise has complex effects on a range of bodily systems involving biochemical, physiological and anatomical processes. There are changes in the use of energy sources, in the blood and muscle chemistry, in the mechanics of heart and lung function, and in the mechanics of musculoskeletal movement. There are differences in these changes according to the type of exercise (aerobic, anaerobic or power training) and there are also long term effects which take place as a function of regular exercise and fitness. As considered in chapters 2 and 3 both exercise and fitness effects may be related to sleep, and it has been hypothesised that SWS is related to both physiological restoration and energy expenditure (Baekeland and Lasky,1966).

This chapter will briefly describe the physiological processes involved in exercise and fitness, with an emphasis on endurance running effects in order to provide a background to the possible effects of this form of exercise on sleep. Important aspects of exercise which may effect sleep include the increased catabolic processes that occur during exercise and the need for later restoration and anabolism, the increased energy consumption of exercise, the increase in body temperature during exercise,and the possible inhibitory influence of exercise on age changes in sleep.

Gross Physiological Responses of Exercise

Exercise begins with changes in heart function and circulation which are initiated in brain centres, probably the cerebral cortex and diencephalon (Astrand and Rodahl, 1977). Heart rate and contractility is increased, metabolic rate is increased, hormones are mobilized and body temperature is elevated. There is also a sympathetic activity increase and a corresponding parasympathetic decrease which results in skeletal muscle receiving an increased share in cardiac output, while at the same time blood flow to the kidneys, skin, and splanchnic areas is reduced, veins become constricted, the pumping action of the working muscle and action of the respiratory movements facilitates the return of blood to the heart in order to make an increased cardiac output possible (Astrand and Rodahl,1977). Blood flow in the working muscle is facilitated by local metabolic changes, and as heat is produced the blood vessels of the skin become dilated to aid in temperature balance within the body (Astrand and Rodahl, 1977). With the beginnings of exercise, pulse rate elevates very rapidly; if exercise is light or moderate a plateau is seen in thirty to sixty seconds and stays relatively constant until exercise finishes (deVries, 1980). If the work load is heavy the heart rate increases until exhaustion sets in. When exercise is complete heart rate decreases rapidly for the first two or three minutes and then decreases more slowly.

Energy Metabolism

Generally physical performance is determined by the individual's capacity for energy output (involving aerobic and anaerobic processes and

oxygen transportation), and their neuromuscular function, joint mobility, and psychological factors (Astrand and Rhodale, 1977). However, at a basic level, exercise is essentially a matter of transforming chemical energy into mechanical energy. In the body this basic set of processes involves the transformation of food (carbohydrates, fats and proteins) into energy available for mechanical use. Both fats and proteins may be converted into carbohydrates, and carbohydrates converted into fats by the processes gluconeogenesis and liponeogenesis respectively. In exercise both fats and carbohydrates are sources of fuel. The use of protein products as fuel has been considered negligible except during periods of extreme shortage of other sources of energy (Astrand and Rodahl, 1977); this conclusion is now coming under some scrutiny (Lemon and Nagle, 1981). At rest, and during light to moderate exercise, fat and carbohydrate contribute in roughly equal proportions to the energy supplies. However as work increases in intensity fat increases its contribution to the energy requirement, and the longer the work lasts the smaller the percentage contribution of carbohydrates (Astrand and Rodahl, 1977). The contribution of carbohydrates is still very important, since at very heavy workloads at above 70 - 80% of an individual's maximum capacity depletion of the muscle glycogen stores may be the factor limiting endurance (De Vries,1980). At a cellular level, in a complex set of processes fats and carbohydrates provide energy for synthesis of the high energy compound adenosine triphosphate (ATP) from its constituents adenosine diphosphate (ADP) and phosphate, producing ATP plus hydrogen (which combines with oxygen to produce water) and carbon dioxide. Thus high energy phosphates are produced from the basic fuel sources. The usual cell functions, such as muscle contraction, use

high energy phosphates and in so doing are split down to ADP and P (a phosphate radical). These processes are reversible and provide a cycle of events within the cell for its operations. Catabolism processes involve breaking down nutrients from their original form into simple end products and releasing energy. Anabolic processes, on the other hand, involve the restorative and synthesizing processes such as building structural storage materials and functional materials from simple materials, and consume energy. During exercise catabolic processes outweigh anabolic processes. This is thought by restorative sleep theorists to be reversed during relaxation and sleep states, such that during sleep much more anabolism takes place (Adam, 1980; Adam and Oswald, 1977; Oswald, 1980).

The rate of energy usage of an organism is called its metabolic rate and is influenced by many factors such as body temperature, environmental temperature, digestion, exercise, and psychological factors such as excitement (Thomas, 1975). During exercise well trained athletes can increase their metabolic rate up to 20 times the basal level (Thomas,1975). Knowing this response to exercise provides an important means of evaluating the effects of the exercise on the body. Measures of O_2 consumption provide an estimate of metabolic rate and the maximal capacity of an individual to consume oxygen (VO_2 max) (Astrand and Rodahl, 1977). In addition such measures provide information about the energy cost of exercise and the potential performance of an endurance athlete.

There are two primary pathways in which energy transformation processes take place:

1. Anaerobic (without oxygen) energy yield occurs when the blood sugars glycogen and glucose are broken down in the absence of oxygen to pyruvic acid and lactic acid with the concomitant release of energy. This is a relatively inefficient process that occurs at times of high energy usage, as in intense exercise such that exhaustion sets in in 1 - 2 minutes or less and results in lactic acid as an end product. When lactic acid concentration becomes too high the muscle cell will cease to function until the lactic acid diffuses into tissues, fluids, and blood, and/or is converted to CO_2 and water (deVries, 1980).

2. Aerobic energy yield is the metabolic process that supplies the energy needs of muscle contraction, in the presence of adequate oxygen, to oxidize the sources of energy completely to carbon dioxide and water. Generally aerobic muscle activity is exercise that is low enough in intensity to be carried on for five minutes or longer. By contrast to anaerobic exhaustion, it is thought that aerobic exhaustion occurs when the cell nutrient supply is depleted (Thomas, 1975). Endurance exercise primarily involves aerobic energy usage and it is conducted for relatively long periods of time. Thus there are low rates of energy usage in endurance runners but the total energy consumption of these athletes is high. The average sedentary 70 kg. man uses perhaps 2500 kcals/day (Astrand and Rodahl, 1977), whereas a 70 kg. distance runner in training might use 3500 or more kcals/day.

Recovery

Very little is known about the recovery processes following exercise expecially endurance running. Only recently Bruce (1984) noted that the recovery processes after marathons had received little attention. After strenuous work there is an energy cost over and above the energy cost of the exercise (deVries, 1980), and metabolic rate remains elevated for several hours, even up to 72 hours (Simonson, 1971, p448). However there are other difficulties in endurance exercise such as muscle soreness, tendon injuries and feelings of fatigue (Bruce, 1984). Tissue difficulties such as delayed muscle soreness are poorly understood and take up to 7 days for recovery (Armstrong,1984). Further there are complex urinary and blood changes which reflect biochemical processes which may take from some hours to up to 3 days to return to normal; Maron and Horvath (1978) indicate these may be related to salt and water retention and negative nitrogen balance similar to that following other stresses such as trauma and surgery. Thus Erickson (1983) points out that up to 5 litres of sweat can be lost during a marathon, placing severe demands on the thermoregulatory and cardiovascular systems. In replacement the athlete should be encouraged to drink (mostly water) in excess of thirst.

Despite the lack of formal scientific work on recovery, the popular running literature discusses recovery in cautious terms, noting that glycogen recovery takes fron 3 - 5 days, and describing the time for full recovery from a marathon as at least a month (eg Higden,1978). Maron and Howarth (1978) comment on the existence of much information on the biochemical aftereffects of marathons as 'colloquial asides' (p146), thus indicating the need for more organized research and information.

Training Effects

The training effects of exercise can be considered at different levels or systems including gross physiological changes, cellular changes and temperature regulation.

The physiological changes brought about by training have been summarized by deVries (1980) as follows:

1. Lowered resting heart rate.
2. Lower heart rate for any submaximal work load.
3 Greater maximal cardiac output.
4. Greater maximal stroke volume.
5. Lower ventilation equivalent that is less ventilation required per unit of oxygen utilized.
6. Greater maximal oxygen consumption.
7. Lower utilization of anaerobic energy sources for a given work load.
8. Capacity for greater oxygen debt.
9. Less displacement of physiological function by any given level of work load and faster recovery to baseline values after completion of exercise.

Further it has been found that more active men have significantly lower systolic and diastolic blood pressure (Montoye, Metzner, Kellner, Johnson and Epstein, 1972). In addition to the changes in cardiovascular function and the efficiency of skeletal muscle cell respiration, training improves glycogen utilization etc. and efficiency of fat use which in turn acts as a carbohydrate sparing process. There is also an increase in tendon, muscle, and bone strength (Appenzeller and Atkinson, 1983a).

In addition to the adaptations that take place in various systems of the body as a function of training there are also changes that take place at a cellular level. Indeed these are the basic changes that result in systems and organ adaptations. There are essentially two types of muscle fibres, fast twitch (FT), which can work intensely and quickly, but can't sustain efficient activity, and slow twitch (ST), which work more slowly and can maintain work for a long period of time. With training there is an increase in muscle protein synthesis, with endurance training in particular causing an increase in the myoglobin and mitochondrial content of muscle cells (de Vries, 1980). This results in an increase in oxygen consumption and allows ST fibres to have increased capacity to use a variety of energy substrates (Lamb, 1984). The changes permit increased oxidation of pyruvate, fat and carbohydrate (Berger, 1982). There is also an increase in the capacity for some FT muscle fibres to engage in oxidative metabolism (de Vries, 1980). In all the changes serve to improve aerobic performance. As skeletal muscle adapts to endurance exercise, it has greater glycogen stores, slower depletion of glycogen, lower lactate levels, and greater oxidation of free fatty acids. It follows that in endurance trained individuals, carbohydrate sparing takes place, and more fat is used for energy than in unfit individuals (Berger, 1982).

The effects of sprint training on anaerobic activity are not as well understood as the effects of endurance training on aerobic metabolism (deVries, 1980). However it has been found that both endurance and sprint training programmes lead to quite similar enzyme adaptations in muscle cells(Hickson, Heusner and Van Huss, 1976). Accordingly, it has

also been found that aerobic training can increase anaerobic capacity in humans (Gollnick, Armstrong, Saltin, Saubert, Sembrovich & Shepherd, 1973).

An important factor in exercise, particularly endurance events, is temperature control. Temperature regulation is located in the hypothalamus, and the process is described as acting very like a themostat in a house, such that, overheating in the hypothalamus (preoptic area) stimulates heat loss through vasodilation of the skin and stimulation of the sweat glands (Brooks and Fahey, 1984). Extended exercise can cause an increase to over 40°C in core body temperature (Brooks and Fahey, 1984). Endurance training improves the tolerance to high temperatures and decreases the temperature regulatory stress, enabling more efficient dissipation of heat and a decrease in the demands of the peripheral circulation (Appenzeller and Atkinson, 1983b).

In sum, training creates a more efficient and stronger organism, which uses energy more effectively, can tolerate the consequent increased body temperatures, and can dissipate the excess heat more effectively.

Hormonal Adaptations to Exercise

Astrand and Rodahl, (1977) note that many hormone producing systems are affected by physical training, but that these changes are not well understood. It appears that many of these training effects are unique for the trained muscles, and not untrained muscles even though the untrained muscles are subject to the same changes in hormone levels (Astrand and Rodahl, 1977). In particular, hormones which regulate the fuel for muscular work, such as cortisol, growth hormone, glucagon and

the catecholamines (the so called stress hormones (Schade, 1983), have all been studied for their effect on metabolism following exercise (Schade, 1983). This group of hormones is particularly important in the maintenance of blood sugars, and play an antagonistic role or counterregulatory role to that of insulin (Schade, 1983). They are also important in providing other fuels for muscle contraction, serving to spare muscle glycogen, and increase exercise tolerance,and, in a delicate balance inhibit the use of glucose by peripheral tissues, and provide adequate glucose for the central nervous system (Schade, 1983). It is inappropriate to consider these processes more than superficially, but some other brief comments on cortisol are important in the context of this thesis.

Cortisol has a number of complex functions which include the mobilization of fatty acids for use as energy, protein mobilization, and a role in the conservation of blood glucose (Brooks and Fahey, 1984). It appears that, in light to moderate exercise, plasma cortisol can show a varying response depending on the degree of stress involved, although, in trained individuals at higher work loads there are lower secretion rates indicating the lower levels of stress involved for these individuals. However, at very heavy work loads, cortisol secretion rates are the same for both trained and untrained subjects. Cortisol secretion rates provide a measure of the physical and psychological stress under which an individual has been placed. The secretion of cortisol is stimulated by all forms of stress, for example physical stress such as exercise or injury, emotional stress such as fear or competition nerves (Berger, 1982), or declining glucose levels (Brooks and Fahey,1984). It is also known that

many other hormones respond to acute exercise (Lamb, 1984), since exercise constitutes a stress which affects a multitude of physiological systems.

Age effects

Ageing is a complex set of processes in which structures change, and cells and tissue function in many organ systems is reduced (Brooks and Fahey, 1984). With the current boom in endurance athletics, the development of veterans events, and the use of exercise as a preventive measure for post coronary patients, the effects of exercise and training on older athletes is of increasing interest. Relatively little has been done in the area, although added interest must have been created by the performance of Lopez in winning the 1984 Olympic Marathon at age 37.

In considering exercise and age effects, there is a need to note that ageing results in a decline in maximal O2 consumption, maximal cardiac output, muscle strength and power, neural function flexibilty, and an increase in body fat (Brooks and Fahey, 1984). Such effects can be reduced by training. The effects of exercise provide a sensitive measure of the physiological decline in ageing (de Vries, 1980), and Bruce (1984) has noted that VO2 max is the best single variable to define the changes in aerobic metabolism and in the cardiovascular system with ageing. The decline in VO2 max over the life span is roughly linear, although those subjects who maintain high activity levels show lower rates of cardiovascular ageing (Bruce, 1984). Thus, in well trained, middle aged individuals, many cardiovascular changes observable in sedentary counterparts do not occur, or progress at a much slower

rate, and, for example, they maintain heart rate and stroke volume at a level consistent with much younger individuals (Wright, Zauner and Cade, 1982) and show shifts in lipoprotein-cholesterol ratios which reflect reduced risk from coronary heart disease (Lobstein, Ismael and El-Naygar,1982). Further, Heath, Hagbery, Ehsani and Holloczy (1981) concluded that veteran athletes seem likely to have a decline of about 5% in VO_2 max per decade compared to about 9% in sedentary men. Other decrements that occur with ageing such as in pulmonary function, in the skeletal system, in joint stiffness, and in flexibility, can also be slowed by regular training (Brooks and Fahey, 1984).

Conclusions

Exercise is not a unitary phenomena, and, depending on the nature and type of exercise, it can have a wide range of physiological and biochemical effects. Exercise involves an increase in metabolic and gross physiological activity with an increase in energy output. However, response to exercise is different for unfit and fit subjects since there are differences in fuel usage, temperature regulation and tissue breakdown during exercise, and differences in recovery from exercise, and in pain and soreness following exercise. In addition exercise results in reduction of the physical decline of ageing.

Each of the foregoing factors may influence sleep, and while the links between exercise physiology and sleep are not well understood, exercise clearly has catabolic and energy consuming effects that provide the basis for the predictions of bodily restorative and energy conservation models of sleep. The discussion in this chapter thus provides a

background of exercise physiology to the three experiments on the effects of aerobic exercise on sleep reported here.

CHAPTER 5

AGE AND SLEEP

CHAPTER 5

AGE AND SLEEP

The experiments reported in this thesis used subjects with a relatively wide age span, from the late teens to the mid fifties. It is therefore important to consider age changes in sleep, particularly across the age ranges of the studies.

The general trends of life span changes in sleep structure and function are known though there are some periods which have been less intensively studied. For example, in the age range from 30 to 50 years there is limited normative data available. In contrast, the sleep of the early years, particularly infancy, but also through adolescence has received the bulk of the research attention. The sleep of the old has also received considerable attention. Nevertheless, there have been only a few studies comparing sleep across age groups (eg Roffwarg et al., 1966; Feinberg, Koresko & Heller, 1967; Feinberg, 1974; Williams et al., 1974; Blois et al., 1983).

In particular there have been two relatively large studies of age and sleep relationships (Williams et al., 1974; Blois et al., 1983). Williams et al (1974) described the EEG characteristics of males and females from birth to old age, using a large number of subjects (247), across 11 age groups. Their work provides the basis of the ensuing conclusions. More recently, Blois et al., (1983) studied normal sleep across 4 age groups aged from 20 to 60 years, but unfortunately the numbers in the older groups were quite small (40-49 n=13: 50-59 n=10), and it is not clear

from their report how many males and females were in each group. An excellent summary of the literature pertaining to life span sleep changes is provided by Spiegel (1981).

While sleep stage changes occur as a function of age, the general cyclical structure remains the same (Feinberg, 1974). Thus there are changes in REM sleep, in SWS (stages 3 and 4),in TST, and in sleep disturbance measures. Each of these will be discussed.

In neonates REM sleep consists of 50 - 80 % (about 8 hours) of TST (Roffwarg et al., 1966), but this percentage reduces to about 30% by the end of the first year. Thereafter there is a slight decline in REM until REM reaches approximately 18% (about 1 hour or less) of TST in the elderly (Roffwarg et al, 1966), although this slight decline in REM in adulthood (20 - 60 years) did not occur in the Blois et al (1983) study.

SWS as a percentage of sleep period time (SPT) progresses gradually within a range of 16-23% from infancy to the 16-19 year period, although with the decline in sleep duration during this period there is a steady decrease in the amount of time in SWS. However, SWS duration begins to decrease more sharply once a subject reaches his 20's, and SWS constitutes on average less than 9% of SPT of a male subject in his 40's (Williams et al., 1974). This decline in SWS in men (based on visual scoring) over the age span of the present work can be seen in Figure 1.

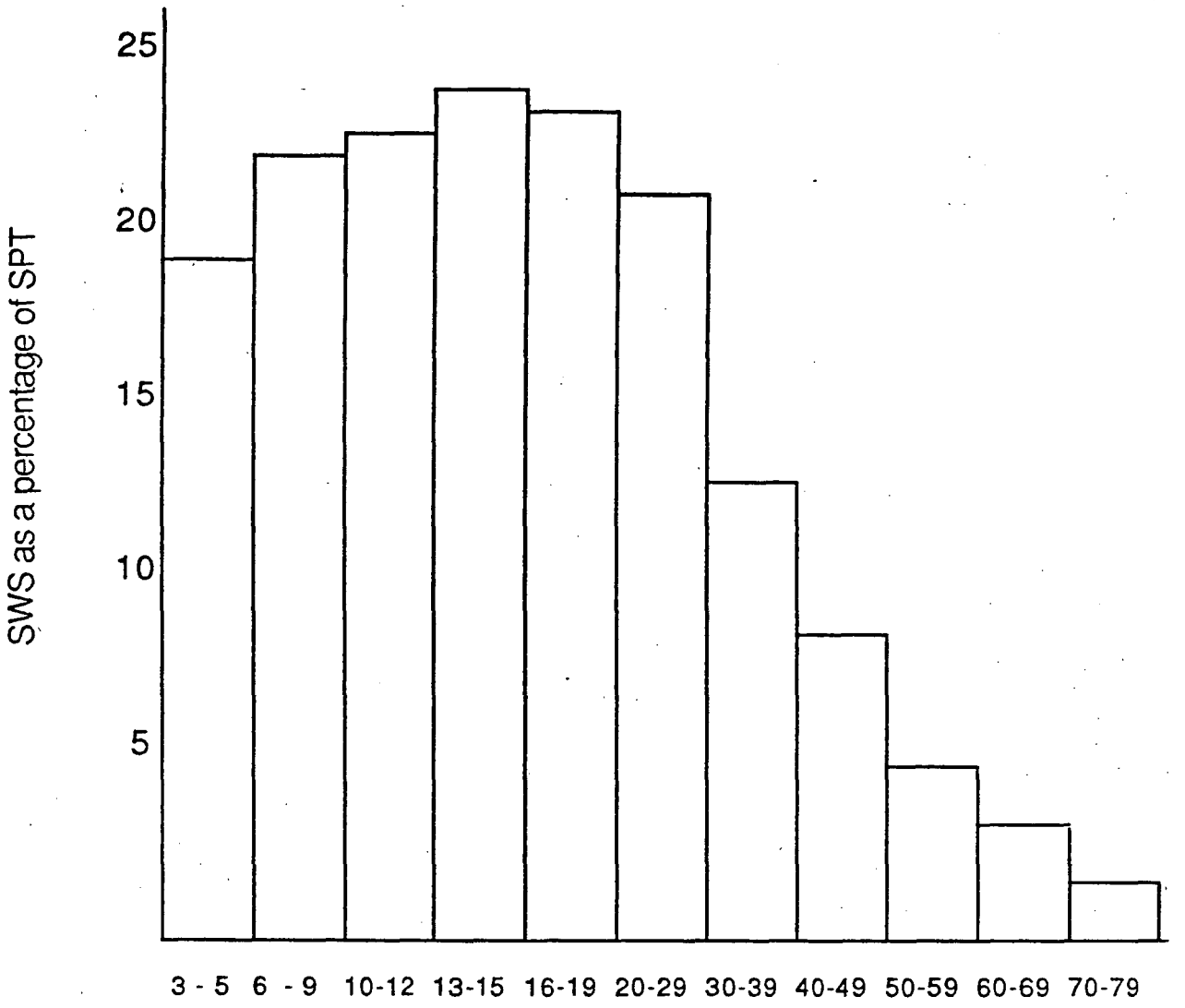

Figure 5.1. SWS as a percentage of SPT in men (Williams et al., 1974)

Measures of sleep duration, time in bed (TIB), SPT, and TST show similar changes in that they are highest in childhood and lowest in adulthood. Thus TST decreases between the fourth and twentieth year from about 10 hours to $7\frac{1}{2}$ hours sleep a night with a slight but gradual diminution of sleep thereafter (Williams et al., 1974).

Sleep disturbances increase with age. Williams et al., (1974) report that sleep efficiency (total sleep time divided by time in bed (TST/TIB)) increases during adulthood as does number of awakenings during the night and number of stage shifts during sleep.

With the increased sleep disturbance with age, it is hardly surprising that it has been suggested that sleep is more easily disturbed by external stimuli. Zepelin (1983) has studied auditory awakening thresholds in young and middle aged adults and reports that the thresholds decline for older subjects. This decline may indicate natural changes in the sleep process, or be a related but secondary process, but, in either case it suggests that sleep diminishes in intensity and becomes more vulnerable to interruption in older subjects (Zepelin, 1983). While Zepelin has found evidence for this phenomenon with auditory awakening thresholds it has yet to be demonstrated that the sleep of older subjects is more likely to be disturbed by other factors, for example, aches and pains, and that this disturbance is a function of changes in sleep or some other related process.

It is clear that the measurement and scoring techniques, and procedures for sleep research can differ from laboratory to laboratory. This means that the available data on sleep and aging is only a guide to

the description of normal sleep for particular ages. Thus the available data indicates the trends between and within sleep stages and cycles across ages. Until there are widely accepted, rigidly structured, and highly reliable measurement and scoring procedures (probably computer conducted), there will need to be internal comparisons in age span studies.

Despite the methodological difficulties, the general effects of age on sleep are known. The cyclical nature of sleep is stable through adulthood although sleep becomes more liable to be fragmented, with REM sleep, TST and SWS gradually diminishing throughout adulthood. Two of these changes, the reductions in sleep duration and SWS, are opposite to the hypothesised effects of exercise. Therefore it might be that exercise would impede the normal ageing effects on sleep. This issue is addressed in later chapters of the thesis.

CHAPTER 6

EXPERIMENT 1

THE EFFECTS OF INTENSITY AND DURATION OF EXERCISE ON THE SLEEP OF FIT MALE SUBJECTS

CHAPTER 6

EXPERIMENT 1

THE EFFECTS OF INTENSITY AND DURATION OF EXERCISE ON THE SLEEP OF FIT MALE SUBJECTS

As reviewed in chapter 3, there are two theoretical views which have traditionally been associated with the prediction that SWS and sleep duration increase as a function of exercise: the energy conservation and bodily restorative theories. However, as is apparent from chapter 2, the results of studies on the sleep of fit subjects have been contradictory.

It has been suggested that where exercise has not resulted in an increase in SWS, the rate of energy expenditure during the exercise has not been high enough (Horne, 1981), and thus the exercise has been insufficiently intense. Of four studies in which intense exercise with high rates of energy expenditure has been used, three have produced facilitative effects of exercise on sleep (Shapiro et al., 1975; Maloletnev et al., 1977, Shapiro et al., 1981), and one failed to produce facilitative effects of exercise (Desjardins et a., 1974). In contrast, three studies have considered exercise of low intensity and long duration (low rates of energy expenditure) (Desjardins et al., 1974; Bonnett, 1980; Buguet et al., 1980) and these studies report no all night facilitative effects of exercise on SWS or sleep duration. Thus the available literature on the effects of intensity and duration of exercise on sleep indicates that duration of exercise of itself is not likely to facilitate sleep, but that exercise of high intensity and of a relatively

long duration produces a facilitative effect on sleep. However no study has attempted to deal with duration and intensity of exercise as independent variables.

In the study to be reported, both the duration and intensity of exercise were varied independently using eleven fit young subjects[1]. It was hypothesized that SWS and sleep duration would increase as a function of increasing intensity (rate of energy expenditure), but not as a function of increasing duration of exercise.

Method

Subjects

Eleven physically fit males with a mean age of 19.5 years were recruited from the university community and a local police academy. Nine subjects were volunteers and two were paid for their participation in the experiment. The subjects were selected on the basis of two criteria. That they had a VO_2 Max as assessed by bicycle ergonometer of greater than 3.7 l/min. (or 52 ml/kg/min. when adjusted for weight) and they were engaged in an ongoing programme of sport which suggested they were capable of performing the required exercise. All subjects were engaged in regular exercise typically including 3 training sessions a week at hockey, football, soccer and rugby. The mean VO_2 max values for the group were 3.94 l/min. and 58.69 ml/kg/min.

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[1]This experiment has been published as experiment 2 in Paxton et al. (1982), a copy of which is included in Appendix 2. When she was under my supervision Miss Jill Newman reported aspects of the experiment in partial fulfilment of an honours degree.

Design

In a repeated measures design four experimental conditions were each run twice in counterbalanced form. The experimental conditions were preceded by a single adaptation night. The four conditions were

1. a no exercise control day,
2. a 1 hour walk at the rate of 5-6 km/hr,
3. a 1 hour run at the rate of 11-13 km/hr and
4. a 6 hour walk at 5-6 km/ hr.

Condition 2 represented a low total work load performed in a relatively short time and at low intensity. Conditions 3 and 4 were designed to represent a high total work load with condition 3 representing a high total work load at high exercise intensity and condition 4 representing a high total work load at low exercise intensity, but of relatively long duration. The total daily energy expenditure of conditions 3 and 4 were designed to be approximately equal. A comparison over conditions 1, 2 and 3 allowed a test of the effects of exercise intensity (no exercise, mild and intense exercise), while a comparison over conditions 1, 2 and 4 allowed a test of exercise duration (no exercise, moderate and long duration).

The order of the four experimental conditions was initially counterbalanced. However it was difficult to schedule some conditions for some subjects. Accordingly the strict counterbalancing was sacrificed in order to avoid prolonged breaks in the experiment. The average order position of the two nights for each condition was 3.6, 4.2, 4.4, and 5.6 for conditions one through four respectively.

Procedures

The subjects were tested for fitness on a bicycle ergonometer. Physical fitness was defined in terms of predicted maximal oxygen uptake (VO_2 max) using the sub maximal exercise nomogram method described by Astrand and Rodahl (1970).

In order to determine the required energy cost of each condition a number of sources were considered to develop estimates of energy expenditure for four broad categories of activity (Altman and Dittmer, 1968; Astrand and Rodahl, 1970; Passmore and Durnin, 1955). They were sleep, 1.17 kcal/min; usual waking activities, 3 kcal/min; walking at 5-6 km/hr, 16 kcal/min; and running at 11-13 km/hr, 16 kcal/min. Energy expenditure during the 24 hours prior to the sleep onset of the four conditions was determined by multiplying the rate of energy expenditure by the time spent in each of the four states (8 hours were assigned to sleep). The resulting values were 3443.6 kcal, 3591.6 kcal, 4221.6 kcal, and 4341.6 kcal for the 24 hour period, covering conditions 1 to 4 respectively. The method is admittedly crude in that it accepted average values for states that covered a wide range of activities, and ignored individual differences. However as the design involved repeated measures, and if it is assumed that variations within each of the four states are not systematically related to conditions, then the method provides sufficient discrimination between conditions for the purposes of the study.

The two high total work load conditions are approximately equated for total energy expenditure and about 25% above control value. This work load appears equivalent to a number of studies some of which have

reported positive effects (eg Zir et al., 1971) and others which have not (eg Bonnet, 1980). In addition the low intensity short duration condition appears equivalent to the exercise load used by Browman (1980) and Horne and Porter (1975), both of which have reported first cycle effects.

Under condition one and in the other conditions during non exercise periods, subjects were instructed to perform only sufficient exercise to complete their daily activities. During the 1 hour walk subjects walked around the university playing fields at the rate of 5-6 km/hr, while during the 1 hour run they covered the same course at 11 - 13 km/hr; both these exercise sessions were conducted between 1600 and 1800 hours. In condition four the subjects walked over a relatively flat nature trail between 1130 and 1800 hours, with a half hour break for lunch at 1300 hours. All exercise sessions were supervised.

The laboratory consisted of two sound attenuated rooms with an adjacent equipment monitoring room. Subjects reported 1 hour before their normal retiring times and prepared for bed. Recording and scoring methods were according to the standardized procedures described by Rechtschaffen and Kales (1968). All recordings were made on a Beckman 411 Dynograph. EEG amplification was set at 100 u V/cm with a time constant of 1 sec. The paper speed was set at 10 mm/sec. The sleep records were scored blind by two scorers who had a page by page interrater agreement of greater than 90% where each page (epoch) accounts for 30 sec. In addition, before lights out a 5-min EKG recording was taken while the subject was awake and in the recumbent position, in order to assess the level of physiological arousal as a

function of exercise condition and thus to have some indication of stress effects.

Subjects were allowed some degree of freedom in selecting the times they came to the laboratory, were allowed to read in bed before lights out if they wished. Lights out was kept to the usual time for each subject, although within the limits of their usual sleep patterns, subjects were able to nominate their time of waking and rising.

All subjects were requested to keep to their regular diet, to avoid excessive amounts of tea or coffee, to not take naps on experimental days, to avoid alcohol consumption and medication during the experiment, and to obtain their normal and regular amounts of sleep during the experiment.

The definition of the sleep variables follows Williams, et al., (1974), with the exception that sleep onset was defined as the first stage 2 epoch. Sleep period time (SPT) was then computed from the first stage 2 until morning wakening. Consequently SPT and TST have a variable relationship to each other depending on the amount of wake time during the night and stage 1 before sleep onset.

Results

The sleep variables analysed included measures of the duration of the lights out period (TIB - time in bed), the time from sleep onset to final awakening (SPT - sleep period time), the total time asleep (TST), the total time awake during TIB (TTA), sleep onset latency (SOL) was the time from lights out to the first stage 2 epoch, wake + movement

time + 1 combined in TIB, minutes and as a percent of TST in each of the sleep stages (MT, 1,2,3,4,3+4 (SWS),2+3+4 (NREM) and REM) and sleep efficiency (TST/TIB). Each variable was analysed using a 4 by 2 ANOVA with repeated measures on each factor. The first factor consisted of the 4 conditions while the second was the conditions replicated. There was no effect of exercise condition on any sleep variable, either over the whole night, or during the first cycle. Table 6.1 shows the results for the basic sleep variables on each experimental night.

As indicated above and in Table 6.1, neither SWS ($F(1,70) = 1.74$, $p>.05$) nor any other NREM variable (Stage 2, $F(1,70) = 2.18$, $p>.05$; Stage 3, $F(1,70) = 1.82$, $p>.05$; Stage 4, $F(1,70 = 0.73$, $p>.05$) was affected significantly by daytime exercise. There was no evidence for an increase in SWS as a function of the intensity or duration of exercise. The mean total night SWS values for the 4 conditions were 80.84, 89.96, 81.14 and 80.11 min. for the no exercise, 1 hour walk, 1 hour run, and 6 hour walk respectively (see Figure 6.1). Similarly the mean TST values for the 4 conditions were 430.6, 438.3, 418.0 and 442.6 min. respectively. The mean values for individual nights on both sets of data are shown in Table 6.1.

There was evidence of continued adaptation to the laboratory during the experiment. Both TTA ($F(1,10) = 6.94$, $p < .05$) and SOL ($F(1,10) = 8.43$, $p < .05$) were significantly decreased from the first

Table 6.1

Mean and standard deviations in minutes of selected sleep variables for fit subjects as a function of the different exercise conditions. Each night in each condition has been given separately.

Variable	Control		One Hour Walk		One Hour Run		Six Hour Walk	
	1	2	1	2	1	2	1	2
SOL	12.1	9.0	10.2	9.6	15.7	9.2	10.2	6.6
	(5.8)	(6.7)	(6.7)	(6.1)	(11.8)	(6.8)	(6.1)	(3.6)
TTA in TIB	14.5	15.0	22.8	13.5	30.7	12.6	12.7	8.7
	(9.6)	(21.6)	(26.9)	(15.8)	(45.3)	(19.6)	(10.7)	(9.6)
Stage 2	217.0	208.9	208.7	215.3	198.5	214.1	232.0	224.2
	(37.3)	(48.0)	(34.8)	(21.4)	(40.1)	(40.5)	(28.8)	(34.8)
Stage 3	31.8	29.7	35.8	29.9	31.0	28.3	27.0	28.5
	(10.0)	(10.0)	(14.0)	(9.9)	(10.9)	(10.4)	(8.9)	(7.4)
Stage 4	51.5	48.6	56.8	57.4	53.2	49.7	54.1	50.5
	(23.8)	(20.8)	(17.0)	(16.6)	(19.7)	(21.8)	(17.4)	(19.0)
SWS (Stage 3+4)	83.4	78.3	92.6	87.3	84.3	78.0	81.2	79.0
	(21.7)	(22.2)	(22.8)	(14.2)	(20.9)	(23.6)	(15.2)	(61.8)
NREM	300.3	287.2	301.3	302.6	282.9	292.1	313.2	303.2
	(23.7)	(49.7)	(33.4)	(20.1)	(27.1)	(32.4)	(19.3)	(32.2)
REM	95.1	91.7	90.8	94.8	83.8	94.6	96.5	96.5
	(31.1)	(29.6)	(23.8)	(27.9)	(22.9)	(24.7)	(13.5)	(19.5)
TIB	450.5	440.2	459.6	453.3	438.3	440.9	459.5	446.1
	(50.9)	(35.2)	(42.2)	(39.8)	(53.3)	(44.8)	(29.6)	(43.8)
TST	436.0	425.2	436.9	439.8	407.6	428.3	446.8	438.3
	(44.5)	(41.3)	(37.0)	(37.4)	(51.5)	(41.6)	(21.7)	(41.6)
SPT	436.1	424.5	446.5	439.6	419.5	432.1	445.6	438.7
	(48.1)	(41.7)	(42.1)	(34.9)	(39.7)	(43.1)	(25.7)	(43.1)
W+MT+1 in TIB	54.7	61.2	67.5	56.0	71.7	54.4	50.6	46.4
	(20.9)	(34.4)	(47.6)	(29.5)	(53.4)	(37.8)	(15.7)	(15.8)

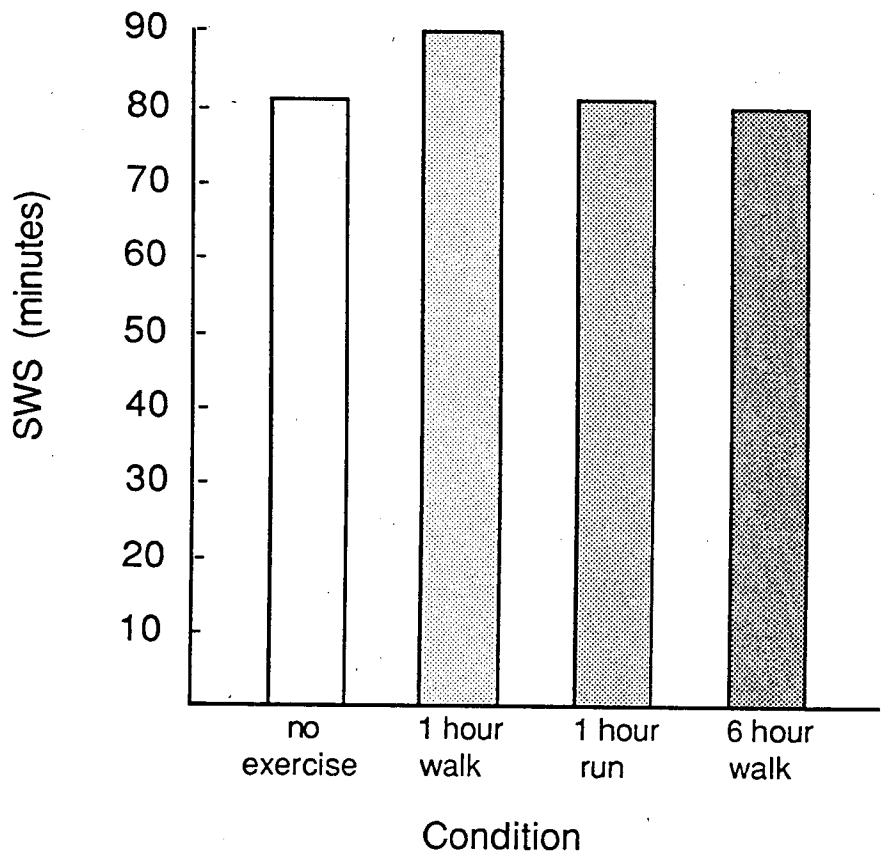

Figure 6.1. SWS (in minutes) in young fit subjects for the four conditions.

to the second occasion of testing for each condition. The greater disturbance on the first testing occasion did not adversely affect SWS as it was on average higher on these nights, though the effect was not significant ($F(1,10) = 4.71, p < .05$). Thus the difficulty experienced in completely counter balancing the conditions would be unlikely to have greatly influenced the effect of the exercise conditions on SWS or sleep duration.

Pre-sleep heart rate (HR) did not differ as a function of either exercise condition ($F(3,30) = 0.7689$, $p>.05$). In addition there was no relationship between HR and SWS or TST within subjects. Thus the change in HR for each subject between the no exercise condition and each of the exercise conditions was not related to the change in SWS or TSTS. This was also true for SWS during the first cycle. The absence of any indication of disturbed sleep, REM sleep changes, or increased presleep heart rate following exercise argues that the failure to observe an exercise effect cannot be attributed to physiological arousal produced by the exercise.

Discussion

This study offers no support for a facilitative effect of exercise irrespective of its intensity or duration, on SWS or sleep duration in fit subjects either over the whole night or in the first sleep cycle. The results are consistent with those studies on fit subjects which show no effect of exercise on SWS and sleep duration.

The failure to find an exercise facilitated increase in these variables has frequently been attributed to a stress factor which counteracts the

effect of exercise (Baekeland and Lasky, 1966; Hauri, 1966; Adamson, et al., 1974; Desjardins et al., 1974; Horne and Porter, 1975; Griffin and Trinder, 1978). The evidence which suggests this possibility consists largely of indications of disturbed sleep following unaccustomed exercise. However this study failed to observe an exercise effect with different levels of exercise and in the absence of physiological arousal.

With one exception in which there was a significant exercise effect for stage 3 of sleep but not for total SWS (Griffin and Trinder, 1978) other studies from our laboratory (Montgomery et al., 1982; Trinder et al, 1982; Paxton et al, 1983) demonstrate consistent results over a range of exercise intensities showing that SWS and sleep duration is independent of day time exercise. One possible explanation for the failure of the subjects in this experiment to show the hypothesized effects is that the amount of exercise remained insufficient. In the present study subjects exercised for an hour at a moderately intense level and while our subjects were a group of talented young athletes, they were not runners, and were not specifically trained for the type of exercise required. Accordingly it is possible that more intense exercise in runners for longer than one hour may produce a SWS effect. The next chapter reports a study which assesses such a possibility.

CHAPTER 7

EXPERIMENT 2

THE EFFECTS OF EXERCISE AND AEROBIC FITNESS ON THE SLEEP OF YOUNG AND OLDER ADULTS

CHAPTER 7

EXPERIMENT 2

THE EFFECTS OF EXERCISE AND AEROBIC FITNESS ON THE SLEEP OF YOUNG AND OLDER ADULTS

Experiment 1 failed to observe an effect of exercise on either SWS or sleep duration despite a variety of exercise conditions. At the time this experiment was being run Horne (1981) had suggested that rate of energy expenditure, over a sufficient duration, was a critical factor in the facilitative effect of exercise on SWS levels. It may have been that condition 3 in experiment 1 was not sufficiently intense to produce this effect. In experiment 2 the intensity of the exercise was increased, and subjects were selected who were capable of the increased level of exercise. Thus the first aim of the experiment was to assess the effect of exercise on sleep using an exercise level at a greater rate of energy expenditure for a longer duration than in experiment 1. In addition several other variables were studied in this experiment.

Aerobic fitness has been shown to be associated with higher levels of SWS (Griffin and Trinder, 1978; Trinder et al., 1982) and longer sleep durations (Montgomery et al., 1982), though the effect does not appear to be due to the aerobic fitness itself (Paxton et al., 1983). Rather the sleep differences may be related to body composition (Paxton et al., 1984a; Paxton et al., 1984b); or type of athletic training (Trinder et al., 1985). The majority of studies reporting differences between fit and unfit groups used young male adults (<25 years of age). Physical training impedes the usual effects of ageing on physiological

physiological attributes such as VO_2 max, maximum cardiac output and muscle strength and power (see Chapter 4). Physical training might also impede ageing effects on sleep. However, the one study which has evaluated the relationship between age and exercise, while observing differences in young subjects, did not find a difference between two slightly older groups (Trinder et al., 1982). Because this study used relatively small samples (n=6), because the age differences between the groups was not that large, and because of the continuing interest in exercise by older individuals, this hypothesis should be further evaluated. Thus the second aim of this study was to assess the effect of physical fitness on sleep as a function of age.

A further finding of Trinder et al., (1982) was that exercise did not affect sleep in the older subjects. The third aim of this study was to assess the effect of intense exercise on both young and older highly trained athletes.

Most studies on the effect of exercise on sleep do not show a positive effect on sleep duration (Desjardins et al., 1974; Horne and Porter, 1976; Walker et al., 1978), though this may be because these studies restricted time in bed. However, in a series of studies in which time in bed was not restricted, Montgomery et al. (1982) also reported that exercise did not effect sleep duration. Nevertheless, as even unrestricted time in bed might be constrained by behavioural patterns, it remains possible that if time in bed were extended subjects may spend more time asleep on nights following afternoon exercise. The fourth aim of this study was to assess the effect of exercise on sleep duration using a procedure in which subjects were confined to bed for a minimum of ten hours each night.

Method

Subjects

Four groups of male subjects, 10 young fit males of average age 22, 12 older fit males of average age 41, 10 young unfit males of average age 22 yrs, and 10 older unfit males of average age 41 years were recruited from the university and general communities. One of the older unfit males was discarded from the data analysis following the scoring of his sleep records when REM sleep onsets were identified. Thus this group was left with 9 subjects. Each of the two groups of young subjects and the two groups of older subjects were equated for age.

The fit subjects had to meet two criteria: They had to reach a predetermined level of cardiovascular fitness as determined by a submaximal test of VO_2 max conducted on a bicycle ergonometer (Astrand and Rhodahl, 1977); and they had to be engaged in regular training. The minimum allowable age adjusted VO_2 max for these groups was set at 3.7 l/min. and 52 ml/kg/min.. All the fit subjects were runners. In one case the subject failed to reach the predetermined VO_2 max levels but the performances of this athlete were of a very high standard suggesting that he was of the type referred to in Astrand and Rodahl (1977), who because of a naturally high heart rate only show their true VO_2 max under a maximal test of VO_2 max. This subject was included in the study. It was impossible to equate the distance run in training by the younger and older subjects since it was necessary to recruit the older subjects from a population of athletes training for long

distance road events while the majority of the younger subjects were preparing for track events and consequently were covering less distance in training.

Unfit physically healthy subjects were selected on the basis of their sedentary life style and non participation in sports or physical activity. The maximum allowable VO_2 max. for these groups was set at 3.0 l./min. and /or an age and weight adjusted 44 ml./kg./min. The average age. weight. aerobic fitness scores and weekly training distance of the groups is shown in Table 7.1.

Design

The design included three variables: age (young versus older). physical fitness (fit versus unfit). and level of exercise (exercise - no exercise). However it was not a complete factorial design as only the fit subjects were required to undergo the exercise. Thus there were four independent groups of subjects; young fit. older fit. young unfit and older unfit. The dependent variables were the amount of SWS. measures of sleep duration and the responses to a series of visual analogue questionnaires about the subjects physical and mental state. In addition a number of other sleep variables were analysed. Following an adaptation night. the sleep of each subject was monitored on two non consecutive periods. Each of the two periods consisted of two consecutive nights in the laboratory.

Table 7.1

Mean age, weight, aerobic fitness scores and weekly training distance for each of the four groups

	Age	Weight in Kgs.	V02 Max. ml/kg/min.	Mean Weekly training distance in Km.
Young Fit n=10	22.2	69.6	68.1	74.4
Young Unfit n=10	22.0	72.7	37.0	-
Older Fit n=12	41.3	63.9	56.9	102.5
Older Unfit n=9	41.5	80.0	31.3	-

In the case of the unfit subjects each two night period comprised two days of their usual sedentary activities. The fit subjects engaged in intense exercise on each day of one experimental period, and did no exercise in the other experimental period. All subjects had their sleep monitored on each of the four nights. In each fit group half the subjects exercised in their first experimental period and half the subjects exercised in the second experimental period. On the exercise days, subjects were required to engage in a strenuous one and a half hour continuous training run during which they covered an average of 20.5 kilometres. The two consecutive days of exercise completed by the fit subjects increases the possibility of build up of exercise induced metabolites and therefore hopefully creating optimum conditions for the increase of SWS and TST. There were two reasons for not requiring the unfit to undergo the exercise condition. First, there has not been an exercise effect shown in unfit subjects in previous studies, and second there was concern over submitting both unfit groups, but particularly the older group, to an extended period of physical stress.

Procedures

On preparation for bed subjects were weighed, and required to fill out questionnaires reporting on subjective state (See appendix 1). This was assessed by a set of visual analogue 100mm scales administered before retiring and on rising on each experimental night. Each subject was required to judge the extent to which they felt physically tired, mentally tired, physically stressed or mentally stressed (not at all - a great deal). On rising they were asked the extent to which they had a

good night's sleep - bad night's sleep, restful night's sleep - disturbed night's sleep, had slept lightly - slept deeply, and the extent to which they felt refreshed - felt fatigued from their night's sleep (see appendix 1).

All sleep recordings were made according to the procedures described in Rechtschaffen and Kales (1968), and scored blind by two trained scorers according to the standard criteria (Rechtschaffen and Kales, 1968), with disagreements being resolved by a third scorer in some cases, and by consultation in others. One exception to these procedures is that a single bipolar electro-oculogram was recorded rather than two monopolar channels (Wells, Allan and Wagman, 1977). The sleep variables analysed were the duration of the lights out period (TIB), the time from sleep onset to final awakening (SPT), the sleep onset latency (SOL), the total time asleep (TST), the total time awake (TTA) during TIB, wake, MT and stage 1 combined in TIB, the minutes in each of the sleep stages (MT, 1, 2, 3, 4, 3+4 (SWS), 2+3+4 (NREM) and REM), minutes in SWS and REM in the first 150 minutes of sleep, the percent of SWS and REM in SPT and sleep efficiency (TST/TIB).

It was not possible to measure accurately the daily energy expenditure of subjects. This does not seem a critical omission since on the basis of the mileage run by the athletic groups there seems no doubt that the average energy expenditure of these groups is substantially above that of the unfit groups.

On sleep assessment nights subjects arrived at the laboratory at 8pm. Following the attachment of electrodes and completion of other

laboratory procedures they were put to bed at approximately 9 pm. Lights out was on request and subjects were permitted to read in bed if they wished. Previously subjects had been asked to sleep as long as they wished. Watches or clocks were not allowed in the bedrooms. Subjects arose in the morning only after they requested to get up, they had been awake for at least 30 minutes after 7 am, or it was 9 am. All subjects had the opportunity to extend the duration of their sleep at either end of the night, and were in bed for a minimum of 10 hours and could sleep for a maximum of 12 hours. A condition of being a subject was that the subject could come into the laboratory in time to be in bed by 9 pm. and that they have no commitments early on the mornings after sleeping in the laboratory. The procedures allowing for extended periods of time in the laboratory were followed in order to provide a better estimation of the total sleep requirements of the subjects than that which is available from procedures which prescribe a constant lights out period.

All subjects were requested to keep to their regular diet, to avoid excessive amounts of tea and coffee, to not take naps during the experimental period, to avoid alcohol consumption and medication, and to obtain their regular amounts of sleep during the experiment, particularly on the night prior to coming into the laboratory. The bedtime and waking time for the night prior to coming into the laboratory was monitored in order to maintain subjects' compliance with this request.

Results

Two sets of analyses were undertaken. First, analyses of variance were conducted on the data from the fit subjects, specifically to test for

exercise and age effects in these samples. The analyses on each variable was a 2 (young fit versus older fit groups) X 2 (exercise versus no-exercise) X 2 (consecutive nights in each condition) ANOVA. Second, analyses of variance were conducted across the four groups on non exercise nights to test for fitness and age effects. The analyses on each variable was a 2 (young versus older subjects) X 2 (fit versus unfit subjects) X 2 (consecutive nights in the no exercise condition) ANOVA. The analyses were conducted on the sleep variables listed above and on the visual analogue scales. Only one of the no exercise conditions for the unfit subjects was used in the second analysis, and the condition used was counterbalanced in order to match the counterbalancing of the fit subjects for exercise and no exercise conditions. Five of the young unfit subjects had their first sequence used and 5 their second sequence of nights used. Because one of the unfit older subjects was discarded only 4 of these subjects had their second sequence of nights used in this analysis, and the other 5 had their first sequence used. In general, the effects of age have been presented in the second analysis.

The Effect of Exercise on Sleep

This set of analyses was to assess the facilitative effects of exercise on sleep, and any differential effects of the exercise on the sleep of the young as opposed to the older fit subjects.

A summary of the results of this analysis are shown in Table 7.2. Exercise did not have a facilitating effect on either SWS or sleep duration (see Figure 7.1). Thus the effect of exercise on minutes in SWS ($F(1,20) = .62$, $p>.10$), Stage 3 ($F(1,20) = 1.77$, $p>.10$), TST

($F(1,20) = .04$, $p>.10$), NREM ($F(1,20) = .80$, $p>.10$), SPT ($F(1,20) = .50$, $p>.10$) TIB ($F(1,20) = .55$, $p>.10$), TST ($F(1,20) = .04$, $p>.10$) and SWS as a percent of TST ($F(1,20) = .72$, $p>.10$) was not significant. However, there was significantly less Stage 4 ($F(1,20) = 6.22$, $p<.05$)and SWS in the first 150 minutes of sleep ($F(1,20) = 4.57$, $p<.05$).

The tendency for exercise to reduce SWS levels was more marked in the younger subjects. The interaction between exercise and age was significant for SWS ($F(1,20) = 4.42$, $p<.05$) and stage 4 ($F(1,20) = 6.22$, $p<.05$), but not for any other variable reflecting SWS, or sleep duration.

The effect of consecutive laboratory nights was to reduce SWS, both minutes ($F(1,10) = 8.33$, $p<.05$) and percent TST ($F(1,20) = 4.44$, $p<.05$), stage 4 ($F(1,20) = 9.86$, $p<.05$), SWS in the first 150 mins ($F(1,20) = 5.96$, $p<.05$) , NREM $F(1,20) = 20.77$, $p<.001$) and TST ($F(1,20) = 10.11$, $p<.01$). Stage 3 (F1,20) = 2,55, $p>.10$), TIB ($F(1,20) = .09$, $p>.10$) and SPT ($F(1,20) = 2.55$, $p>.10$) were not different. The reduction in SWS on the second night was more marked in the exercise condition though the interaction between exercise and first versus second night was only significant for minutes in SWS ($F(1,20) = 5.89$, $p<.05$). The exercise related reduction in SWS tended to be greater in the younger subjects with the three way interaction approaching significance ($F(1,20) = 3.17$, $.10>p>.05$).

Table 7.2

Duration in minutes of selected sleep variables for fit subjects as a function of age and exercise. The data for each of the two consecutive nights in each condition has been given separately.

Variable	Younger (N=10)				Older (N=12)				Significant Effect
	No Exercise		Exercise		No Exercise		Exercise		
	1	2	1	2	1	2	1	2	
TIB	596.8	614.9	620.0	612.2	583.0	556.4	574.8	579.6	AEN*
TST	542.4	557.9	566.2	535.4	527.7	502.4	532.3	496.0	N*
SPT	566.1	584.4	597.3	631.4	559.9	522.4	549.5	542.6	
SOL	12.1	21.7	16.4	28.5	12.7	14.6	15.1	28.7	E*,N*
TTA in TIB	54.4	57.1	53.8	76.8	55.3	54.0	42.5	83.5	N*, EN*
W+MT+1 in TIB	113.0	126.2	122.2	148.9	122.0	124.8	114.3	165.8	E*,N**,EN*
Sleep Efficiency	.909	.907	.913	.875	.905	.903	.926	.856	N*
Stage 2	276.5	280.3	286.0	287.7	296.1	277.3	301.9	276.9	
Stage 3	31.0	37.4	39.3	32.4	29.4	26.9	32.1	28.8	
Stage 4	57.9	48.8	52.9	38.4	14.5	12.6	15.8	11.7	A*,E*,N**,AE*
Stage 3+4	88.9	86.2	92.2	70.8	43.9	39.5	47.9	40.5	A*,*N**,AE*,EN*
3+4 in 1st 150 mins	61.2	56.2	55.9	48.2	31.8	27.8	30.3	25.3	A**,E*.N*
Stage 2+3+4	365.4	366.5	378.2	358.5	340.0	316.8	349.8	317.4	N**
REM	119.1	123.0	118.6	105.3	121.0	114.8	110.7	96.2	E*
REM in 1st 150 mins	8.4	13.5	9.2	9.0	14.9	14.5	10.5	11.8	E*

A = Age; E = Exercise; N = Nights; * = significant < .05; ** = significant <.01; Sleep variables are defined in the text. Standard deviations for these figures are presented in Appendix 3.

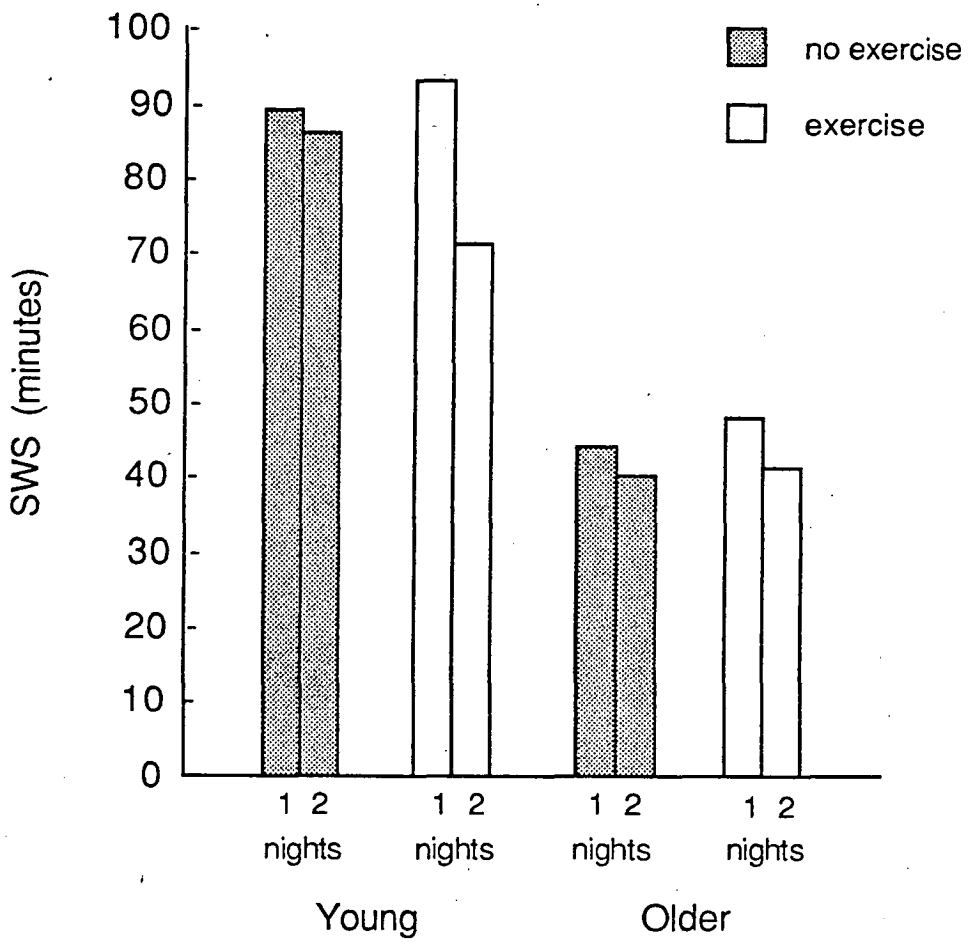

Figure 7.1. SWS (in minutes) in the young and older fit subjects following both no exercise and exercise conditions on each of the two nights.

The three way interaction was significant for TIB ($F(1,20) = 4.54$, $p<.05$) such that TIB was lower on night two than night one following exercise as compared to non exercise for the younger subjects, with the inverse pattern for the older subjects. Sleep period time approached significance ($F(1,20) = 3.31$, $.10>p>.05$) while TST was not significant ($F(1,20) = .34$, $p>.10$).

In summary there was no evidence for a facilitative effect of exercise on either SWS or sleep duration. Indeed there was evidence that some aspects of SWS were reduced by the exercise; an effect more marked in the younger subjects. Slow wave sleep and TST were reduced on the second of the consecutive nights. This is likely to be in part due to the elevated sleep duration achieved in this study. Time in bed, SPT and TST were all at least 100 minutes greater than in previous studies from this laboratory (Montgomery et al, 1982). In addition, the exercise by nights interaction suggests that the accumulated effects of two strenuous training sessions may also have contributed to a reduction in SWS on the second night.

The later result raises the possibility that a facilitative effect of exercise might only be found on the first night. Inspection of Table 7.2 indicates a small effect in this direction. However, an anlysis using only the first of each of the consecutive two nights shows that while the exercise effect for minutes in stage 3 was significant ($F(1,20) = 6.72$, $p<.05$), stage 4 showed a non significant change in the other direction ($F(1,20) = .42$, $p.>10$) and the difference in SWS was not significant ($F(1,20) = 2.29$, $p<.10$). No age by exercise interaction effects were significant. Thus this analysis provides no evidence to support the hypothesis that exercise increases SWS levels in fit subjects.

The duration of REM sleep, REM as a percentage of TST, and REM in the first 150 minutes were all reduced following exercise ($F(1,20) = 9.69$, $p<.01$; $F(1,20) = 15.6$, $p<.001$; $F(1,20) = 6.56$, $p<.05$ respectively). No other effects involving REM sleep were significant. Sleep onset was longer following exercise ($F(1,20) = 6.45$, $p<.05$) and on the second of the two consecutive nights ($F(1,20) = 11.12$, $p<.01$). The pattern of results suggests that two factors influenced sleep. First, exercise had a disruptive effect, as indicated by reduced SWS and REM, and longer SOL. Second, the long sleep durations on the first of the two consecutive nights affected sleep on the second night. These two influences are also apparent in measures of disturbed sleep. Thus the amount of wake and disturbed sleep (W + MT + 1) during TIB was greater following exercise ($F(1,20) = 4.76$, $p<.05$), though this difference was not significant for wake during TIB ($F(1,20) = 1.45$, $p>.10$), or for sleep efficiency ($F(1,20) = 1.36$, $p>.10$). However, there was substantial disturbance on the second exercise night with the exercise by nights interaction being significant for wake during TIB ($F(1,20) = 4.54$, $p<.05$), W + MT + 1 during TIB ($F(1,20) = 4.58$, p,.05) and nearly so for sleep efficiency ($F(1,20) = 3.88$, $.10>p>.05$). Sleep disruption was greater on the second night of each condition ($F(1,20) = 6.07$, $p<.05$); $F(1,20) = 10.09$, $p<.01$; $F(1,20) = 7.21$, $p<.05$) for TTA in TIB, W + MT + 1 in TIB and sleep efficiency respectively).

Questionnaires administered each evening indicated that all subjects were more physically tired ($F(1,20) = 51.88$, $p<.001$) and physically stressed ($F(1,20) = 26.79$, $p<.001$) following exercise. The physical

stress was more marked on the second exercise night ($F(1,20) = 14.00$, $p<.001$). Both age groups were more mentally tired following exercise ($F(1,20) = 7.23$, $p<.05$) while the older subjects reported greater mental stress ($F(1,20) = 4.93$, $p<.05$).

The morning questionnaire was consistent with the sleep recordings in showing some evidence of disturbed sleep following exercise. Subjects reported significantly more restlessness during sleep ($F(1,20) = 13.45$, $p<.01$). They also reported poorer and less refreshing sleep, though these differences were not quite significant ($F(1,20) = 3.15$, $.10>p>.05$; $F(1,20) = 3.51$, $.10>p>.05$ respectively). Also subjects reported sleeping less deeply on the second night following exercise ($F(1,20) = 5.05$, $p<.05$).

Fitness and Age Effects

A summary of this data is shown in Table 7.3. As was hypothesized SWS levels were elevated in the fit subjects (see Figure 7.2). Minutes of SWS ($F(1,37) = 5.08$, $p<.05$), percentage SWS ($F(1,37) = 4.15$, $p<.05$) and stage 3 ($F(1,37) = 4.28$, $p<.05$) were all significantly elevated, while stage 4 showed a non significant increase ($F(1,37) = 2.87$, $.10>.p>.05$). As would be expected the main effect of age was also significant for SWS in minutes ($F(1,37) = 53.52$, $p<.001$) and percent SWS ($F(1,37) = 41.24$, $p<.001$), stage 4 ($F(1,37) = 71.29$), $p<.001$) and stage 3 ($F(1,37) = 6.97$), $p<.05$). However most importantly, the fitness by age interaction was not significant indicating that both age groups showed the fitness effect on SWS to an equivalent degree ($F(1,37) = 0.38$, $p>.10$; $F(1,37)=1.07$, $p>.10$; $F(1,37)=.04$, $p>.10$;

$F(1,37)=.85$, $p>.10$) minutes and percent SWS, stage 4 and stage 3 respectively).

Sleep duration did not show a significant effect of fitness. While the fit subjects had longer TIB, SPT, TST and NREM sleep, the analysis just failed to reach significance in each case ($F(1,37) = 3.61$, $.10>p>.05$; $F(1,37) = 3.29$, $.10>p>.05$; $F(1,37) = 3.06$, $.10>p>.05$; $F(1,37) = 3.58$, $10>p>.05$ respectively). However SOL was shorter in the fit subjects ($F(1,37) = 6.73$, $p<.05$). Total sleep time and NREM sleep were significantly longer in younger subjects ($F(1,37) = 5.67$, $p<.05$; $F(1,37) = 7.00$, $p<.05$ respectively, but not TIB, SPT, or SOL ($F(1,37) = 3.09$, $10>p>.05$; $F(1,37) = 3.43$, $10>p>.05$); $F(1,37) = 3.19$, $10>p>.05$) respectively). Again the fitness by age interactions were not significant ($F(1,37) = .00$, $p>.10$; $F(1,37) = .00$, $p>.10$; $F(1,37) = .09$, $p>.10$; $F(1,37) = .03$, $p>.10$; $F(1,37) = .84$, $p>.10$ for TIB, SPT, TST, NREM sleep and SOL respectively).

The second of the two consecutive nights was lower for SPT ($F(1,37) = 4.13$, $p<.05$), TST ($F(1,37) = 5.84$, $p<.05$) and NREM sleep ($F(1,37) = 8.26$, $p<.05$), but was not significantly different for TIB ($F(1,37) = 2.54$, $p>.10$), SOL ($F1,37) = 1.14$, $p>.10$), or any SWS variable ($F(1,37) = 1.01$, $p>.10$; $F(1,37) = .03$, $p>.10$; $F(1,37) = 2.70$, $p>.10$; $F(1,37) = .30$, $p>.10$ for minutes and percent SWS, stage 4 and stage 3 respectively).

Table 7.3

Duration in minutes of selected sleep variables for non exercise nights as a function of age and level of fitness. The data for each of the two consecutive nights in each condition has been given separately.

Variable	Younger				Older				Significant Effect
	Fit (N=10)		Unfit (N=10)		Fit (N=12)		Unfit (9)		
	1	2	1	2	1	2	1	2	
TIB	596.8	614.9	592.1	554.1	583.0	556.4	544.0	523.8	
TST	542.4	557.9	544.7	507.4	527.7	502.4	497.9	469.9	A*N*
SPT	566.1	584.4	562.8	521.1	559.9	522.4	517.8	495.9	N*
SOL	12.1	21.7	28.7	30.7	12.7	14.6	21.7	17.9	F*
TTA in TIB	54.4	57.1	47.5	46.7	55.3	54.0	46.1	53.8	
W+MT+1 in TIB	113.0	126.2	111.1	105.8	122.0	124.8	117.4	138.6	AFN*
Sleep Efficiency	.909	.907	.920	.916	.905	.903	.915	.897	
Stage 2	276.5	280.3	277.7	248.4	296.1	277.3	296.8	274.2	N*
Stage 3	31.0	37.4	31.0	29.4	29.4	26.9	16.8	18.2	A*F*
Stage 4	57.9	48.8	46.2	46.0	14.5	12.6	6.1	3.3	A*
Stage 3+4	88.9	86.2	77.2	75.4	43.9	39.5	22.9	21.5	A**,F*
3+4 in 1st 150 mins	61.2	56.2	49.4	52.2	31.8	27.8	17.9	17.1	A**,F*
Stage 2+3+4	365.4	366.5	354.9	323.8	340.0	316.8	319.7	295.7	A*,N*
REM	119.1	123.0	127.5	125.4	121.0	114.8	106.8	86.7	A*
REM in 1st 150 mins	8.4	13.5	13.5	13.3	14.9	14.5	22.1	13.8	A*

A = Age; F = Fitness; N = Nights; * = significant < .05; ** = significant <.01; Sleep variables are defined in the text. Standard deviations for these figures are presented in Appendix 3.

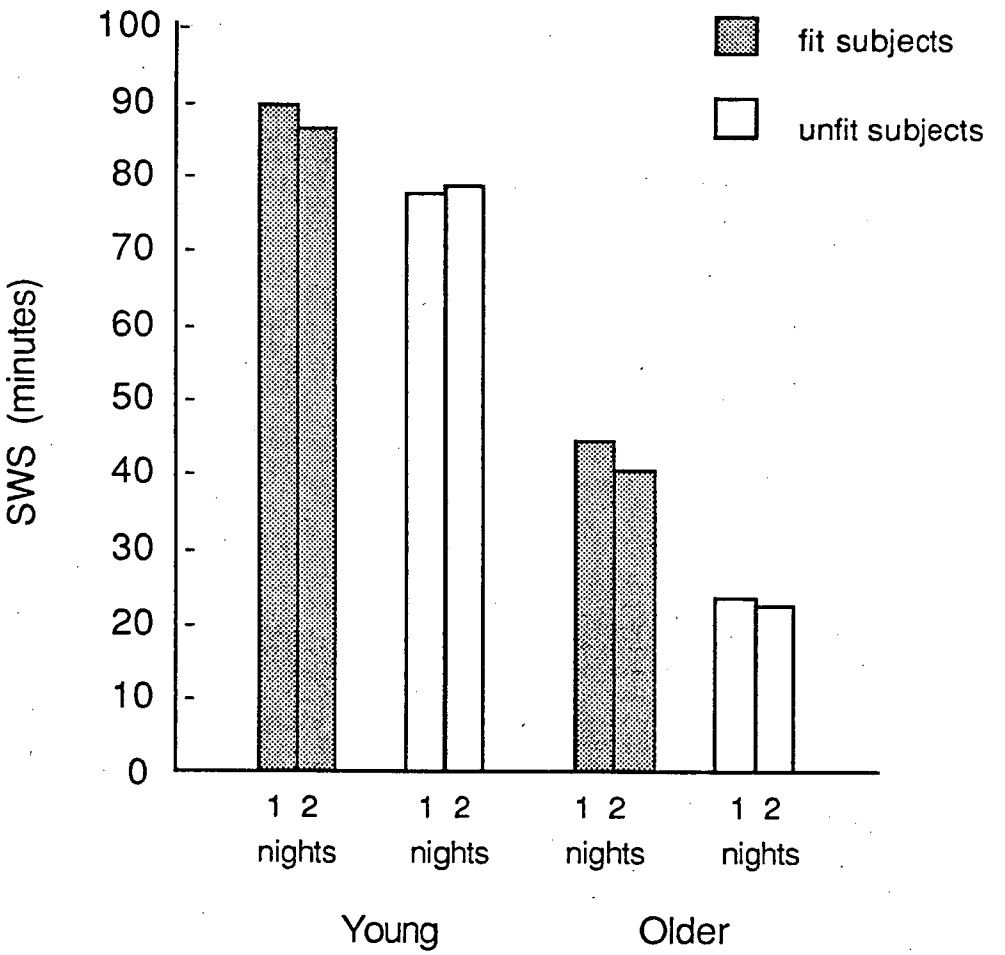

Figure 7.2. SWS (in minutes) in both young and older, fit and unfit subjects, on each of the two nights.

REM sleep was generally unaffected by fitness though the young unfit subjects had a higher proportion of REM than the other three groups. Thus the fitness by age interaction was significant for percentage REM ($F(1,37) = 4.65$, $p<.05$). REM in minutes was higher in the younger subjects ($F(1,37) = 5.58$, $p<.05$), though this is probably reflected in the higher TST in these subjects as REM percent did not differ as a function of age ($F(1,37) = 1.57$, $p>.10$). Finally REM in the first 150 minutes was higher in the older subjects, possibly as a consequence of the lower SWS levels ($F(1,37) = 4.09$, $p<.05$).

Measures of sleep disturbance were generally not significant. The only exception was a fitness by age by nights interaction for W + MT + 1 during TIB ($F(1,37) = 4.13$, $p<.05$).

On the evening questionnaires the fit subjects reported less physical stress ($F(1,37) = 4.86$, $p<.05$), particularly on the second night ($F(1,37) = 5.53$, $p<.05$), and marginally less physical tiredness ($F(1,37) = 3.07$, $10>p>.05$). Mental tiredness and stress were not related to fitness. In contrast the older subjects reported greater mental tiredness and stress ($F(1,37) = 5.10$, $p<.05$; $F(1,37) = 6.67$, $p<.05$ respectively), but age was not related to physical tiredness or stress. Mental tiredness was greater on the first of the two nights ($F(1,37) = 4.13$, $p<.05$).

Finally, only one comparison for the morning questionnaires was significant; that was the fitness by age interaction for the scale, "Do you feel refreshed from your night's sleep?". Young fit and old unfit subjects reported their sleep to be more refreshing than the other two groups of subjects ($F(1,37) = 5.43$, $p<.05$).

Discussion

The data from this experiment clearly failed to show a facilitative effect of afternoon exercise on either SWS, or sleep duration, during the following night. The negative result was obtained despite using very fit subjects and intense exercise with high rates of energy expenditure in both young and an older group of subjects. Further , the failure to observe the sleep duration effect was obtained despite ample opportunity for subjects to extend their sleep following exercise. The results are unlikely to have been due to low statistical power as this study used considerably more subjects than any previous study testing this hypothesis. Moreover, the data is consistent with the majority of reports in the literature (see chapter 2).

The results of both objective and subjective measures indicate that under certain circumstances exercise disrupts sleep. There was also evidence that, as part of a general disruption of sleep, exercise may reduce SWS levels. The disruption was most marked following the second of the two consecutive exercise sessions and presumably reflects the accumulated effect of the two hard training runs.

The increased time in bed resulted in an increase in sleep duration. Possibly as a consequence of this the pattern of sleep altered on the second of the two consecutive nights. However, the effect of consecutive nights was most marked in the analysis of the fit subjects, and was difficult to disentangle from the effects of consecutive intense exercise sessions (see table 7.2). The analysis of the no exercise nights

showed only a reduction in stage 2, total NREM and sleep duration on night two (see table 7.3). The failure to observe a facilitative effect of exercise was not due to either extended sleep, or consecutive nights, or their combination, as an analysis of night one was also negative.

SWS, but not sleep duration, was elevated, in both fit groups in this study, though the measures of sleep duration each approached significance. Also sleep latency was reduced in these subjects . The results with the older group contradicts the findings of an earlier study (Trinder et al., 1982). However the earlier study used athletes from various sports, the majority of which had a high anaerobic training component. The subjects in the present study were all endurance runners. A comparison of the younger aerobically trained athletes in this study with aerobically trained and power trained athletes, has shown the aerobically trained group to have the highest levels of SWS (Trinder et al., 1985). The present results for the older group, when compared to the results of Trinder et al., (1982) extended that finding to an older sample. Thus the results indicated that aerobically trained runners have higher levels of SWS, and possibly higher sleep durations, than sedentary control subjects irrespective of the age of the subjects. The present data do not reveal the mechanism involved in this difference.

In regard to the sleep onset data from this experiment it should be noted that sleep onset data are usually highly skewed, and the data from this experiment and the two others in this thesis are not exceptional. Skewed data violates the assumption in the analysis of variance of normal distributions. Nevertheless it has been standard practise among sleep researchers to ignore this violation because the analysis of variance is a

robust procedure and the degree of error involved "does not constitute a serious problem" (Keppel, 1982, p.86).

In general the results of this experiment confirm and extend to an older group the findings of previous experiments from this laboratory (Paxton et al., 1982; Montgomery et al., 1982). Exercise did not have an effect on either SWS or sleep duration, while aerobic fitness is associated with increased SWS, reduced sleep onset latency, and possibly increased sleep duration. This pattern of results might be interpreted as indicating that habitual exercise results in adaptations in sleep consistent with restorative and energy conservation theories. However, as the effect appears linked to aerobic training, this interpretation is unlikely to be correct.

The first two studies have assessed the effects of exercise on sleep over a range of exercise levels. However it remains possible that even more extreme intensity and duration of exercise such as that encountered in a marathon could produce an exercise effect. The next chapter reports a study in which the sleep of subjects is studied after they have completed a 42.2 kilometre marathon.

CHAPTER 8

SLEEP DISRUPTION FOLLOWING A MARATHON

CHAPTER 8

SLEEP DISRUPTION FOLLOWING A MARATHON

The studies reported in the two previous chapters have failed to show that exercise has a facilitative effect on either SWS or sleep duration in fit subjects. The exercise conditions used in these experiments was mild to intense where severe exercise was a 1½ hour run of an average distance of 20.5 kilometres. It remains possible that the exercise effect only occurs as a result of even more intense exercise in fit subjects such as would occur in the completion of a standard marathon. The most direct and substantial evidence supporting this latter proposal derives from Shapiro et al. (1981).

Their study used a sample of 6 young distance runners with a mean age of 21.7 years who completed a 92 kilometre marathon. The results showed a marked increase in SWS and an increase in TST on the first night following the marathon.

The experiment reported in the last chapter (chapter 7) found that exercise can result in sleep disturbance. This effect occurred with both young and older male subjects, and is consistent with previous findings in young subjects (Baekeland and Lasky, 1966; Buguet et al., 1980; Shapiro et al., 1975; Shapiro et al., 1981). In two of these experiments (Shapiro et al., 1975; Shapiro et al., 1981) sleep disturbance was related to intense exercise. It has been shown (Buguet et al, 1980) that stress as indicated by elevated cortisol levels is found in subjects whose sleep was disturbed. It therefore seems possible that sleep disruption can be a consequence of exercise induced stress.

The aim of the final experiment was to determine the effect of a marathon on sleep duration, SWS and measures of disturbed sleep[2]. In addition, the effect of the marathon on cortisol levels was measured to determine the relationship between exercise induced stress and sleep.

Method

Subjects and Design

Eight runners of average age 40.75 years slept in the sleep laboratory on 8 occasions: an adaptation night to allow for possible first night effects, two consecutive nights, each following a 90 minute hard training run between 1600 and 1800 hours in the evening, two consecutive nights on non training days, and the three nights following a standard 42.2 kilometre marathon. On non training days the subjects were asked not to engage in exercise or strenuous activity, and on the hard training days subjects were required to engage in a strenuous workout and covered at least 18 kilometres. The order of the three conditions was counterbalanced across subjects. All subjects were experienced distance athletes in full training, and 7 of them had completed at least three marathons. The averaged age adjusted oxygen uptake value of the subjects was 58.75 ml/kg/min. The age of the sample reflects the age and experience of competing marathon runners in Tasmania. These subjects were also part of the older fit sample in the previous experiment (chapter 7) and thus the no exercise and 90 minute run condition was part of the data for that analysis.

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[2]This experiment has been published as Montgomery et al. (1985) a copy of which is included in Appendix 2.

The sample was drawn from entrants in three marathons run over an 8 month period in June, August and January of 1982 and 1983 respectively, at different places in Tasmania, with different starting times: 11 am., 9.30 am., and 8 am. respectively. The runners required between 2 hours 32 minutes and 3 hours 9 minutes to complete the events, with an average time of 2 hours 51 minutes.

Assessment Procedures

On preparation for bed subjects were weighed, and required to fill out questionnaires reporting subjective state (see appendix 1). This was assessed by a set of visual analogue 100 mm. scales administered before retiring and on rising on each experimental night. Each subject was required to judge the extent to which they felt physically tired, mentally tired, physically stressed and mentally stressed (not at all - a great deal) on retiring. On rising they were asked the extent to which they had a good night's sleep (good night's sleep - bad night's sleep), a restful night's sleep (restful sleep - disturbed sleep), had slept deeply (slept lightly -slept deeply), and the extent to which they felt refreshed from their night's sleep (feel refreshed - feel fatigue).

Core body temperature data was also collected immediately before retiring. Subjects were required to take a temperature reading using a rectal thermometer inserted to a depth of 5 cm.

All sleep recordings were made according to the procedures described in Rechschaffen and Kales (1968) and were scored blind by two trained scorers according to standard criterion (Rechschaffen and Kales, 1968). Scoring disagreements were resolved by discussion. One

exception to these procedures is that a single bipolar electro-oculogram was recorded rather than two unipolar channels (Wells, Allan and Wagman, 1977). The sleep variables analysed were the duration of the lights out period (TIB), the time from sleep onset to final awakening (SPT), the sleep onset latency (SOL), the total time asleep (TST), the total time awake (TTA) during TIB, wake + MT + 1 combined in TIB, the minutes in each of the sleep stages (MT, 1, 2, 3, 4, 3+4 (SWS), 2+3+4 (NREM), and REM, minutes in SWS and REM in the first 150 minutes of sleep, the percent of SWS and REM in TIB, and sleep efficiency (TST/TIB).

On sleep assessment nights subjects arrived at the laboratory at 8 pm. Following the attachment of electrodes and completion of other laboratory procedures they were put to bed at approximately 9 pm. Lights out was on request and subjects were permitted to read in bed if they wished. Previously subjects had been asked to sleep as long as they wished. Watches and clocks were not allowed in the bedrooms. Subjects arose in the morning only after they requested to get up, they had been awake for at least 30 minutes after 7 am, or it was 9 am. All subjects had the opportunity to extend the duration of their sleep at either end of the night, and were in bed for a minimum of 10 hours and could sleep for a maxmimum of 12 hours. A condition of being a subject was that the subject could come into the laboratory in time to be in bed by 9 pm. and that they have no commitments early on the mornings after sleeping in the laboratory. The procedures allowing for extended periods of time in the laboratory were followed in order to provide a better estimation of the total sleep requirements of the subjects than that which is available from procedures which prescribe a constant lights out period.

All subjects were requested to keep to their regular diet, to avoid excessive amounts of tea and coffee, to not take naps during the experimental period, to avoid alcohol consumption and medication, and to obtain their regular amounts of sleep during the experiment, particularly on the night prior to coming into the laboratory. The bedtime and waking time for the night prior to coming into the laboratory was monitored in order to maintain subjects' compliance with this request.

In addition to sleep data, urine produced during the experimental periods was collected for analysis of various metabolites. In this case we were interested in cortisol secretion rates as a indicator of stress. The urine collection periods were from time of arising (7.00 am) in the morning to 3 pm, 3 pm to lights out and from lights out to time of rising except on the day of the marathon when the collection period was from rising in the morning of the marathon to the period immediately before the marathon, from immediately before the marathon to lights out, and from lights out to the following morning. Cortisol was measured as urinary free cortisol by radioimmunoassay, using the Amerlex Cortisol RIA Kit from the Radiochemical Centre, Amersham, England.

Results

For the analysis the data for each of the two days of the no exercise and the exercise conditions was averaged, and in a planned comparisons ANOVA these two conditions were compared. The sleep of subjects was similar under these two conditions, and following this comparison, the total data for the conditions was combined and compared to the data from the marathon condition day 1. The marathon

day 1 data was also compared to the marathon day 2 and marathon day 3 data. The sleep variables analysed included measures of total sleep duration, disturbed sleep, and each stage of sleep expressed in minutes and as a percentage of total sleep time. In the case of the visual analogue scales the number of mm. from the left hand end of the scale to the subjects mark were used. This data was then analysed using the procedures used for the sleep variables. The urine for each cortisol collection period was analysed and converted into a secretion rate of nanomols per hour and also analysed using a planned comparisons ANOVA.

As can be seen from Table 8.1 sleep was dramatically disturbed on marathon night 1 compared to training and non training nights, and marathon nights 2 and 3. Total sleep time was significantly decreased on marathon night 1 relative to each of the other conditions (TST $F(1,7) = 8.92$, $p<.01$). Further, REM latency was significantly longer ($F(1,7) = 6.94$, $p<.05$), and total REM ($F(1,7) = 5.89$, $p<.05$), REM in the first 150 minutes ($F(1,7) = 5.72$, $p<.05$),and a sleep efficiency index (TST/TIB) ($F(1,7) = 6.55$, $p<.05$) were all significantly reduced. In 7 subjects SWS was decreased (see Figure 8.1), and total time awake was increased, results which failed to reach significance (SWS ($F(1,7) = 3.512$, $p>.05$); TTA ($F(1,7) = 3.41$, $p>.05$), and in 6 subjects there was an increase in SOL ($F(1,7) = 2.76$, $p>.05$), though again this was not significant. Sleep had returned to normal with no significant rebound (with the exception of REM latency) by the second post-marathon night.

Table 8.1

Mean and standard deviation values in minutes of selected sleep variables during each experimental condition

Variable	No exercise	Exercise	Mean of No exercise and exercise	Marathon 1	Marathon 2	Marathon 3
Sleep Onset Latency	11.3 (7.8)	14.7 (9.5)	13.0 (8.0)	21.8 (11.7)	11.0* (7.0)	11.9* (8.2)
TTA	39.9 (35.6)	46.1 (26.7)	43.0 (30.3)	72.6 (50.6)	36.3 (18.7)	38.1 (27.7)
TST/TIB	.92 (0.6)	.92 (.04)	92* (.05)	.84 (.10)	.92* (0.04)	.93* (.05)
REM latency	74.0 (18.1)	88.5 (29.3)	81.3*+ (16.9)	138.6 (62.8)	66.3* (22.0)	81.2 (32.3)
SWS latency	17.9 (5.3)	19.8 (9.4)	18.8 (6.8)	19.7 (7.2)	21.1 (6.5)	26.9 (18.3)
REM in the 1st 150 min	14.2 (8.0)	11.4 (5.0)	12.8* (6.6)	6.1 (8.5)	14.4* (8.7)	13.4 (9.8)
SWS in the 1st 150 min	33.2 (16.3)	30.3 (11.3)	31.7 (13.1)	25.6 (13.4)	30.9 (19.1)	24.9 (15.3)
SWS	42.2 (18.6)	46.4 (14.9)	44.3 (16.5)	36.3 (19.4)	42.8 (32.5)	39.5 (25.1)
REM	98.8 (23.3)	92.5 (19.1)	95.6* (21.0)	66.8 (22.7)	92.0 (27.1)	108.1* (30.8)
TST	445.2 (32.6)	456.0 (24.9)	450.6* (27.8)	387.1 (45.0)	445.9* (50.4)	455.6 (31.8)

* Indicates significant comparison against Marathon 1 at $p < .05$
** Indicates significant comparison against Marathon 1 at $p < .01$
\+ Indicates significant comparison between mean of no exercise and 90 minute run and marathon 2 at

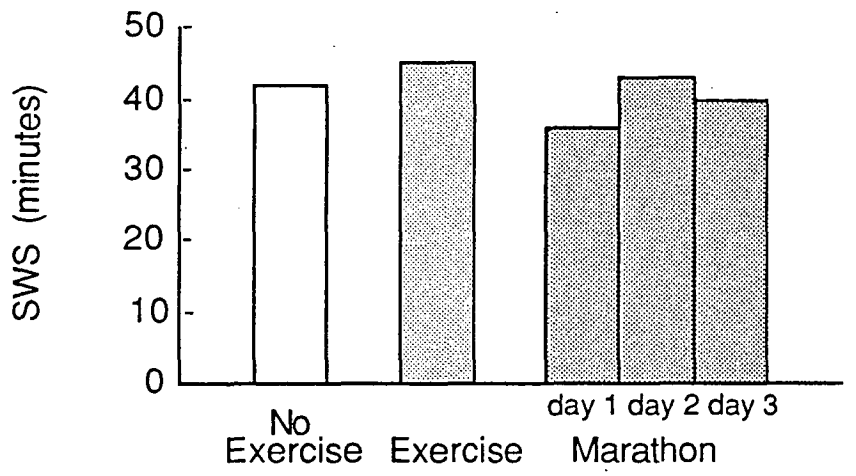

Figure 8.1. SWS for the no exercise and 90 minute run conditions, and marathon days 1, 2, and 3.

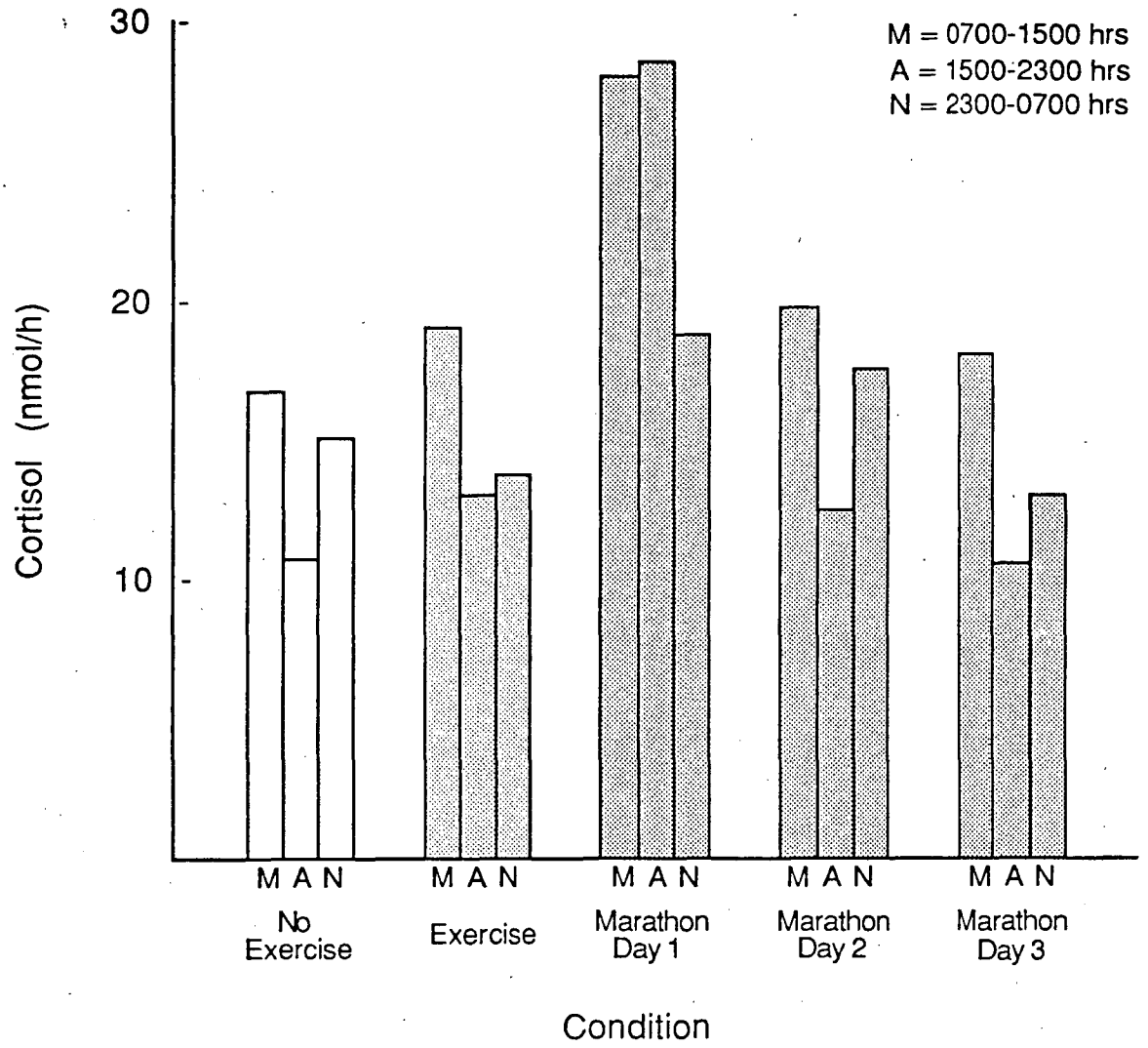

Figure 8.2. Cortisol secretion rates for the no exercise and 90 minute run conditions, and marathon days 1, 2 and 3.

The questionnaire data is consistent with the sleep data. Thus subjects report being significantly more physically tired ($F(1,7) = 19.35$, $p<.01$) and physically stressed ($F(1,7) = 18.21$, $p<.01$), and report their sleep to be more disturbed on the marathon night 1 ($F(1,7) = 19.04$, $p<.01$). Finally, urinary cortisol output per hour was significantly higher in the period immediately prior to the marathon, the marathon and post marathon period, and the first post marathon night, when compared to post marathon days 2 and 3 ($F(1,7) = 7.80$, $p<.01$), and exercise and non exercise conditions ($F(1,7) = 7.47$, $p<.01$). As shown by comparison of Figures 8.1 and 8.2 the increase in cortisol output rates occuring on marathon day 1 appears to coincide with the decrease in total SWS in minutes, and the return to normal of the cortisol output rates is also consistent with the return to normal of the SWS.

The core body temperature data suggested an increase in temperature on the marathon night 1 (mean of exercise and no exercise night, 36.40 degrees centigrade, mean for marathon night 1, 36.65 degrees centigrade). However this was not a central feature of the experiment, and although complete data was available on 5 subjects, some unreliability in the data collection due to other demands on the subjects lead to this data not being pursued vigorously.

Discussion

The results indicate that older fit subjects do not show a facilitatory effect on SWS or sleep duration of the very intense exercise of a competitive marathon. However they are consistent with other studies which show sleep disturbance following exercise (and in particular

disturbance in REM sleep). The stress of the exercise was apparent in both hormonal and subjective measures, and both electrophysiological and subjective estimates of sleep indicated profound sleep disturbance.

The results are at variance with the study of Shapiro et al (1981) which found a facilitation of SWS, TST and SOL after exercise in fit young subjects. Further, in a study reported after the commencement of this experiment Torsvall, Akerstadt and Lindbeck (1984) also failed to gain facilitative effects of similar intensive exercise on SWS using a sample aged between 30 and 35 years. It thus appears unlikely that the facilitative effects of exercise extends to an older population. Further it is not clear from our studies under what conditions SWS would be facilitated by exercise since we have failed to find such a facilitative effect on fit young subjects under a variety of levels of exercise. The range of exercise we have used includes mild exercise such as a one hour walk, to the strenuous one and a half hour run and a marathon. This suggests that the exercise effect on SWS is not directly related to exercise but to some other factor or factors.

It should be noted that there is no rebound effect of SWS on marathon days 2 and 3. This also gives credence to the view that SWS levels appear to be determined by metabolic and constitutional factors which reflect a longer time scale than the bodily restorative processes involved in day to day activities. It has also been suggesed that there is a negative relationship between adrenocorticosteroid levels and both REM and SWS (Gillin, Jacobs, Snyder, and Henkin, 1974; Buguet et al, 1980; Bunnell et al, 1980; Weitzman, Zimmerman, Czeisler, and Ronda, 1981). In our data the increase in cortisol levels on marathon day 1 coincide

with some slight, though consistent, decrease in SWS, a significant decrease in REM sleep and an increase in REM latency. When cortisol levels had returned to baseline levels the sleep variables had also returned to baseline, or had rebounded in the case of REM latency. The increase in cortisol secretion rates occurred throughout the marathon day. The fact that it occurred in the period prior to the marathon indicates a strong psychological component in this stress response.

An alternative explanation of the REM suppression effects of intense exercise is that it may be a consequence of elevated body temperature. The poikilothermic properties of REM sleep are thought to result in REM inhibition when ambient or hypothermic temperatures are outside thermic neutrality (Parmeggiani, 1977, 1980). The same mechanism may operate to reduce REM time during exercise induced elevations in body temperature. In the present study some body temperature data was collected. However, the measurements were not pursued vigorously because of the demands made on subjects for the collection of other data. The data available indicated elevated rectal temperatures on the marathon night.

The finding in several previous papers that REM sleep was reduced and REM latency increased in conjunction with an increase in SWS (Bunnell et al., 1980, Shapiro et al., 1975, Shapiro et al., 1981) suggested that the REM effect was a consequence of the increase in SWS. That is, because SWS occurs predominantly early in the night and early in the sleep cycle, the SWS increase may have prevented the appearance of REM sleep until further on into the night. The present data suggests that both REM disturbance and SWS decrease are a direct

result of the stress of the intense exercise. This interpretation is consistent with other studies which report REM inhibition following intense exercise in the absence of a SWS increase (Bonnet,1980; Buguet et al.,1980; Torsvall,et al., 1984). It is possible the REM inhibition is a more sensitive index of exercise induced stress, occuring at less intense exercise levels than the inhibition of SWS.

In none of the three experiments reported in this thesis was there a sleep facilitation effect of exercise. However, the previous finding that SWS is related to fitness has been replicated in young subjects, and extended to older subjects. The implications of the results of these experiments are considered in the next chapter.

CHAPTER 9

DISCUSSIONS AND CONCLUSIONS

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DISCUSSION AND CONCLUSIONS

Two aspects of the effects of exercise on sleep have been considered in the three experiments in this thesis. They are the immediate effects of an exercise session on sleep (the exercise effect), and the effects of regular exercise on sleep (the fitness effect). In no experiment was there an exercise facilitation effect on sleep duration or SWS. In contrast to the failure to find an exercise effect, the results of experiment 2 replicated previous findings of a fitness facilitation effect on sleep in young subjects, and extended this finding to an older age group. The implications of these findings will now be considered.

The Exercise Effect

An increase in SWS and sleep duration (the more general summary term will be used throughout this chapter) following exercise has only been reported in fit subjects, and then only occasionally. This led to the hypothesis that the exercise effect depended upon a level of exercise which was greater than unfit subjects could undertake. However experiment 1 failed to establish the effect in fit subjects following exercise ranging in severity from a 1 hours walk to a 1 hours run. It had been suggested that in addition to the amount of exercise, the rate at which it was performed was relevant. Thus it was hypothesized that it had to be performed at high rates of energy expenditure (Horne, 1981). This hypothesis was further tested in experiments 2 and 3. Despite the use of very fit subjects, and two levels of severe exercise, a ninety minute run (experiment 2) and both a ninety minute run and a 42.2 kilometre race (experiment 3) the effect did not occur.

It is possible that an exercise-stress effect disturbs sleep and prevents the occurrence of an exercise-facilitation effect on sleep. This seems unlikely since under conditions of exercise where stress was not apparent (experiment 1), an exercise effect did not occur, and neither SWS nor sleep duration were elevated on the second and third nights following a marathon, despite cortisol levels returning to baseline values.

The failure to establish an exercise effect under the stringent exercise conditions of experiments 2 and 3 indicates that it is probably the effect of strenuous exercise interacting with variables yet to be determined that is important in the facilitative effect of exercise on SWS and sleep duration.

Two aspects of work on the effects of exercise on sleep may cast light on the process. First, Horne (1981) has proposed that an increase in SWS following exercise is a consequence of body temperature control processes, and in a series of experiments has shown an increase in SWS following conditions of passive body heating (Horne and Reid, 1984; Horne and Shackel, 1985), and following exercise induced body temperature increase (Horne and Staffe, 1983; Horne and Moore, 1985). If body temperature control processes are involved it may explain the fickle nature of the exercise effect following exercise in the unfit since they are unlikely to become hot enough via exercise to call such temperature control processes into use (Horne, 1981). It is more difficult to explain the failure to get an exercise effect in fit subjects under the intense exercise conditions of experiments 2 and 3. However, Horne (1981) suggests that the effect is less likely to be obtained if the exercise is conducted under cool conditions. He suggests this occurs particularly

where there is cooling of the face (cf. Horne and Moore, 1985). Hobart has a temperate climate with average daily maximums of 21.6 degrees centigrade in February, and 11.8 degrees centigrade in July. Thus it is possible that climatic factors account for the failure to observe an exercise effect in these studies.

However it seems likely that the subjects did get sufficiently high levels of exercise to induce a body temperature increase in experiment 3 since all subjects were in a competitive marathon and performed at a high level. In this experiment core body temperature was measured in five subjects, and elevated rectal temperatures occurred on the first post marathon night. Horne and Shackell (1985) reported that prolonged passive heating given too close to bed time disrupts rather than facilitates sleep. Thus the extended intense exercise in experiment 3 may have produced a similar prolonged heating effect. Nevertheless the work of Horne (Horne and Staffe, 1983; Horne and Moore, 1985; Horne and Reid, 1985; Horne and Shackell, 1985) consistently produces an increase in SWS in fit and unfit subjects under passive (hot baths), and active (strenuous exercise) heating conditions. He has argued that the critical factor is an increase in brain temperature during the heating phase and a subsequent reduction by bed time. Horne's studies were run under controlled wind and temperature conditions, whereas the exercise of experiments 1, 2 and 3 were conducted under a variety of wind and temperature conditions though with cool temperatures predominanting. Thus it is possible that the studies reported here have managed to produce either too much or insufficient heating.

A second aspect of the effects of exercise on sleep is the suggestion that women appear to more easily show the effect (Bunnell et

al., 1983; Trinder, personal communication, see Chapter 2). If this is so it may also be related to temperature regulation since women tend to rely on slightly different mechanisms to achieve the same thermoregulatory control (Brooks and Fahey, 1984). Further analysis of exercise - sleep effects comparing men and women may elucidate the processes involved in any sleep changes.

Generally SWS and sleep duration have been discussed together as variables that were predicted to change following exercise. It was therefore important in these experiments to maximize the possibility of the occurence of an increase in TST following exercise (see Montgomery et al., 1982), and experiments 2 and 3 made substantial allowance for this possibility. However sleep duration did not increase following exercise and is consistent with the difficulty in experimentally producing increases in SWS. Indeed, these experiments indicate that SWS and sleep duration are more likely to be disturbed, rather than facilitated, by the physical stress of exercise.

Exercise induced sleep disturbance affected both REM sleep and, to a lesser extent, SWS in both experiments 2 and 3. This may be related to the stress of the exercise since, as has been discussed in experiment 3, there is increasing evidence that there is a negative relationship between corticosteriod levels and both SWS and REM sleep (Buguet et al., 1980; Bunnell et al., 1983; Weitzman, et al., 1981; Gillin, et al., 1974), and the decreases in REM sleep and SWS appear to relate to changes in cortisol levels in experiment 3.

It appears that REM sleep is more responsive to exercise induced stress than SWS, and may be a more sensitive index of this process. It

is possible, however, that there are other factors operating on both REM and SWS. One alternative explanation for the decrease in REM sleep discussed in experiment 3 is that it may be a consequence of elevated body temperature. If this is the case then REM sleep may be decreased first, by body temperature increase, and then SWS may be affected. The process could be even more complex since Horne & Shackell (1985) have suggested that SWS increase might follow a subsequent decrease in body temperature.

Both bodily restorative and energy conservation theories predict that sleep duration and SWS increases as a result of exercise, although energy conservation theory limits the prediction to conditions of a net decrease in energy resources. However the three experiments here failed to establish an exercise facilitation effect on any sleep variable. Thus the hypothesis that both SWS and sleep duration are responsive to changing levels of day time catabolism and energy expenditure is not supported by these experiments.

The Fitness Effect

In experiment 2 fit subjects had more SWS and a tendency to have more sleep than unfit subjects. This was in contrast to Trinder et al (1982) who established a fitness effect in young subjects but not in a sample of older fit subjects. The difference is most likely a function of the difference in the type of training the older subjects undertook. The present subjects were aerobically trained distance athletes as compared to the mixed aerobic and anaerobic training undertaken by Trinder's (Trinder et al., 1982) subjects, and it has recently been shown

by Trinder et al. (1985) that athletes who engaged in different types of physical training have different sleep architectures. They reported that an aerobically trained group of subjects had more SWS, longer sleep and shorter SOL's than subjects who emphasized mixed aerobic and anaerobic, or power training.

It is noteworthy that the effect of fitness was consistent over age. Thus age and fitness did not interact to result in a less severe diminution in (or more youthful) sleep structure as might be predicted by analogy from the effects of exercise on cardiovascular function. This suggests that the development of fitness at any age will result in an increase in SWS. Such an hypothesis is contrary to the finding of Paxton et al., (1983) who failed to show a change in SWS as a function of getting fit. However, this difference is also likely to be due to the type of training used. Her subjects engaged in a variety of aerobic and anaerobic exercises, and not the purely aerobic training of the subjects in experiments 2 and 3. Thus the fitness effect is a phenomena which extends across age groups and which appears to be a function of aerobic training.

Nothwithstanding the previous comments Paxton et al.'s (1983) data would suggest that any changes in sleep as a function of getting fit occur only slowly over months and years, since their subjects showed no changes in SWS following a mean fitness training period of 3.6 months. Such an hypothesis is consistent with the suggestion that the changes to sleep following metabolic changes occur only slowly (Dunleavy et. al., 1974; Montgomery et al., 1982). Further it is consistent with lay views about the development of fitness for many sports in which it is assumed

that competitive fitness takes some years to develop. Thus if an increase in fitness leads to an increase in SWS then the time scale during which an increase in fitness might affect sleep is problematic and requires investigation.

Theoretical and Methodological Implications

The bodily restorative view holds that an increase in both SWS and sleep duration are indicative of increased bodily restoration. This should occur after the tissue breakdown of an exercise session. However, the failure to find an exercise facilitation of SWS and sleep duration does not offer support for this view. While the present version of this theory does not account for the results, a change in the theory in which more intense restoration takes place across the same duration of sleep, or same amount of SWS, could accommodate the results. Two possible arguments along these lines are as follows. First, it might be that sleep duration and SWS does not change as a function of need for restoration, but rather indicates the period of time during which restorative processes are particularly active. Thus the relative levels of restorative activity are not reflected in the duration of sleep and SWS but are reflected in other variables yet to be described. Second, SWS and perhaps other aspects of sleep might vary not in duration but in intensity following a need for restoration. A similar argument applies to an energy conservation view. Thus sleep duration and SWS might not increase in duration following exercise but there might be a marked decrease in metabolic processes during sleep and SWS. Such possibilities would require more sophisticated scoring procedures than are regularly available at present (Horne, 1981; Torsvall et al., 1984). One attempt at such scoring

procedures in exercise-sleep studies has been the work of Torsvall et al., (1984) who used computer analysis to assess the power density function of the various sleep stages. Torsvall et al (1984) found a modest increase in power density of SWS following exercise. This effect should be replicated.

Restorative views also have difficulty accounting for the recent data from Horne's laboratory (Horne and Staffe, 1983; Horne and Moore, 1985; Horne and Reid, 1985; Horne and Shackell, 1985). Horne has shown an increase in SWS following both hot baths and an exercise induced body temperature rise. Further, SWS changes may also be subject to modification by sex, climatic conditions, and the intensity and time of day of the exercise. Thus there are a number of factors which may influence SWS only some of which are directly related to catabolic activity or energy expenditure. It therefore seemes unlikely that a single set of processes such as restoration or energy conservation will account for exercise facilitation of SWS.

The energy conservation view offers three possible hypotheses: that species differences in sleep are a function of species differences in achieving energy balance, and these are in part a function of their activity level; that individual differences in sleep within a species are a function of long term energy balance and thus in part on long term activity levels; and that within species differences in sleep are a function of short term changes in activity levels. The fitness effect data from experiment 2 suggests support for the second of these hypotheses and indicates an empirical relationship between SWS and sleep duration, and long term exercise patterns. The data are also consistent with the data

reported by Montgomery et al., (1982), and with the view that sleep variables are slow to respond to changes in metabolic factors (Dunleavy et al., 1974). Of course, it might also be that bodily restorative processes only influence sleep over the long term, and a restorative view could also accommodate the fitness effect with such an argument. However the fact that the results of the fitness effect analysis in experiment 2 apply to aerobic fitness in endurance athletes, and not to other forms of fitness (cf. Trinder et al., 1985) appears to preclude interpretations along these lines.

It is not clear what the mechanisms might be in an aerobic fitness effect on SWS, but there are however a number of methodological issues which bear on the fitness - SWS and TST relationship, including the assessment of type and amount of food intake, type of exercise, and energy expenditure during and after exercise. Each of these variables might need careful consideration in subsequent experiments, and analysis of their effects may elucidate the mechanisms of aerobic fitness - sleep relationships.

While restorative and energy conservation theories have predicted that SWS and sleep duration will increase following exercise, the empirical literature has frequently produced results inconsistent with these predictions. In the case of energy conservation theory this may be because compensatory caloric intake has not been controlled. Nevertheless, the present data does not support traditional views. Instead it indicates that fitness and high rates of energy expenditure are not sufficient to produce the exercise facilitation effect on sleep. Further it shows that high rates of energy expenditure over a long duration can disrupt sleep.

The present data adds further weight to the evidence against the traditional statements of the restorative and energy conservation theories, as these views require an increase in SWS and sleep duration following exercise. However the failure to find support for these theoretical views does not mean that in general they are wrong, as the views do not have to specify that sleep is responsive to exercise for it to be energy conserving or restorative. Further the duration of SWS and sleep duration may not change to accommodate restorative or energy conservation functions but rather may reflect conditions during which restorative or energy conservation processes occur.

Thus the conditions under which exercise facilitates sleep have not yet been clearly delineated. In addition intensive exercise of long duration can severely disrupt sleep, and aerobic training appears related to increased SWS and sleep duration. Adequate explanation of these phenomena is awaited.

REFERENCES

Adam, K. (1980). Sleep as a restorative process and a theory to explain why. Progress in Brain Research, 53, 289-305.

Adam, K. and Oswald, I. (1977). Sleep is for tissue restoration. Journal of the Royal College of Physicians. 11, 367-388.

Adam, K. and Oswald, I. (1983). Protein synthesis, bodily renewal and the sleep-wake cycle. Clinical Science, 65, 561-567.

Adamson, L., Hunter, W.M., Ogunremi, O.O., Oswald, I. and Percy-Robb, I.W. (1974). Growth Hormone increase during sleep after daytime exercise. Journal of Endocrinology, 62, 473-478.

Allison, T., and Van Twyver, H. (1970). The evolution of sleep. Natural History, 79, 56-65.

Altman, P.L., and Dittmar, D.S. (Eds.), (1968). Metabolism. Maryland: Federation of American Societies for Experimental Biology.

Appenzeller, O. and Atkinson, R. (1983,a). Nutrition for physical performance. In Appenzeller, O and Atkinson, R. (Eds.) Sports Medicine: Fitness, Training, Injuries. 2nd. Ed. Baltimore-Munich: Urban and Schwarzenberg. 57-90.

Appenzeller, O. and Atkinson, R. (1983,b). Temperature regulation and sports. In Appenzeller, O. and Atkinson, R. Sports medicine: Fitness, Training, Injuries. 2nd Ed. Baltimore - Munich: Urban and Schwarzenberg. 11-33.

Armstrong, R.B. (1984). Mechanisms of exercise-induced delayed onset muscular soreness: A brief review. Medicine and Science in Sports and Exercise, 16, 529-538.

Aserinsky, E. (1969). The maximal capacity for sleep: Rapid eye movement density as an index of sleep satiety. Biological Psychiatry, 1, 147-159.

Astrand, P. and Rodahl, K. (1977). Textbook of Work Physiology. New York:McGraw-Hill Book Co.

Atkinson, D.E. (1968). The energy charge of the adenylate pool as a regulatory parameter. Interaction with feedback modifiers. Biochemistry, 7, 4030-4034.

Atkinson, D.E. (1970). Enzymes as control elements in metabolic regulation. In Boyer, P. D. (Ed). The Enzymes (Vol 1). New York, Academic Press.

Baekeland, F. (1970). Exercise deprivation, sleep and physiological reactions. Archives of General Psychiatry, 22, 365-369.

Baekeland, F. and Lasky, R. (1966). Exercise and sleep patterns in college athletes. Perceptual and Motor Skills, 23, 1203-1207.

Berger, R.A. (1982). Applied Exercise Physiology. Philadelphia, Lea and Febiger.

Berger, R.J. (1975). Bioenergetic functions of sleep and activity rhythms and their possible relevance to aging. Federation Proceedings, 34, 97-102.

Berger, R.J. (1984) Slow wave sleep, shallow topor and hibernation: Homologous states of diminished metabolism and body temperature. Biological Psychology, 19, 305-326.

Blois, R., Feinberg, I., Gaillard, J.M., Kupfer, D.J., and Webb, W.B. (1983). Sleep in normal and pathological aging. Experientia, 39, 551-558.

Bonnet, M.H. (1980). Sleep, performance and mood after the energy expenditure equivalent of 40 hours of sleep deprivation. Psychophysiology, 17, 56-63.

Brebbia, D.R. and Altschuler, K.Z. (1965). Oxygen consumption and electroencephalographic stage of sleep. Science, 150, 1621-1623.

Brooks, G.A. and Fahey, T.D. (1984). Exercise Physiology: Human bioenergetics and its applications. New York: John Wiley and Sons.

Browman, C.P. (1980). Sleep following sustained exercise. Psychophysiology. 17, 577-580.

Browman, C.P. and Tepas, D.I. (1976). The effects of pre-sleep activity on all night sleep. Psychophysiology, 13, 536-540.

Bruce, R.A. (1984). Exercise, functional aerobic capacity, and aging - another viewpoint. Medicine and Science in Sports and Exercise,16,8-13.

Buguet, A., Roussel, B., Angus, R., Sabiston, B., and Radomski, M. (1980). Human sleep and adrenal individual reactions to exercilse. Electroencephalography and Clinical Neurophysiology, 49, 515- 523.

Bunnell, E.B., Bevier, W. and Horvath S.M. (1983). Effects of exhaustive exercise on the sleep of men and women. Psychophysiology, 20, 50-58.

Clugson, G.A. and Garlick, P.J. (1982). The response of protein and energy metabolism to food intake in lean and obese man. Human Nutrition: Clinical Nutrition, 36C, 57-70.

Desjardins, J., Healey, T., and Broughton, R. (1974). Early evening exercise and sleep. Sleep Research. 3,31.

deVries, H.A. (1980). Physiology of Exercise. Dubuque, Iowa: Wm. C. Brown Company.

Drucker-Colin (1979). Protein molecules and the regulation of REM sleep: Implications for function. In Drucker-Colin, R., Shkurovick, M. and Sterman, M. (Eds). The Functions of Sleep. New York, Academic Press, 99-112.

Dunleavy, D.L., Oswald, I., Brown, P. and Strong, J.A. (1974). Hyperthyroidism, sleep and growth hormone. Electroencephalography and Clinical Neurophysiology, 36, 259-263.

Eastman, C.I.and Rechtschaffen, A. (1982). Effect of thyroxine on sleep in the rat. Sleep, 2, 215-232.

Erickson, D.G. (1983). Fluid and Fluid Electrolytes in Endurance training. In Appenzeller, O. & Atkinson, R. Sports Medicine: Fitness, Training, Injuries. 2nd. Ed. Baltimore-Munich: Urban & Schwarzenberg, 157-171.

Feinberg, I. (1974). Changes in sleep cycle patterns with age. Journal of Psychiatric Research, 10, 283-306.

Feinberg, I. and Carlson, V.R. (1968). Sleep variables as a function of age in man. Archives of General Psychiatry, 18, 239-250.

Feinberg, I., Koresko, R.L. and Heller, N. (1967). EEG sleep patterns as a function of normal and pathological aging in man. Journal of Psychiatric Research, 5, 107144.

Fisher, C. (1965) Psychoanalytic implications of recent research on sleep and dreaming. 1. Empirical findings. Journal of the American Psychoanalytical Association, 13, 197-270.

Garlick, P.J., Clugston, G.A., Swick, R.W. and Waterloo, J.C. (1980). Diurnal pattern of protein and energy metabolism in man. American Journal of Clinical Nutrition, 33, 1983-1986.

Gillin, J.C., Jacobs, L.S., Snyder, F., and Henkin, R.I. (1974). Effects of ACTH on the sleep of normal subjects and patients with Addison's disease. Neuroendocrinology, 15, 21-31.

Griffin, S.J., and Trinder, J. (1978). Physical fitness, exercise a human sleep. Psychophysiology, 15, 447-450.

Gollnick, P.D., Armstrong, R.B., Saltin, B., Saubert, C. W., Sembrowich, W.L.and Shepherd, R.E. (1973). Effect of Training on Enzyme activity and Fibre composition of Human Skeletal Muscle. Journal Of Applied Physiology, 34, 107-111.

Hartman, E.L. (1973) The functions of sleep. New Haven, Yale University Press.

Hauri, P. (1968). Effects of evening activity on early night sleep. Psychophysiology, 4, 267 - 277.

Heath, G.W., Hagberg, J.M., Ehsani, A. and Hollszy, J.O. (1981). A Physiological Comparison of young and older endurance athletes.

Journal of Applied Physiology : Respiration, Environment, Exercise physiology, 51, 634-640.

Hickson, R.C., Heusner, W.W., and Van Huss, W.D. (1976). Skeletal muscle enzyme alterations after sprint and endurance training. Journal of Applied Physiology, 40, 868-872.

Higden H. (1978). How not to run it. The 10 most common mistakes made by new marathoners. In Henderson, J. (Ed). The Complete Marathoner. World Pulications : Mountain View, CA, 113-116.

Horne, J.A. (1977) Factors relating to energy conservation during sleep in mammals. Physiological Psychology, 5, 403-408.

Horne, J.A. (1978). A review of the biological effects of total sleep deprivation. Biological Psychology, 7, 55-102.

Horne, J.A. (1979). Restitution and human sleep: A critical review. Physiological Psychology, 7, 115-125.

Horne, J.A. (1980). Sleep and body restitution. Experientia, 36, 11-13.

Horne, J.A. (1981). The effects of exercise on sleep: A critical review. Biological Psychology, 12, 241-290.

Horne, J.A. (1983). Human sleep and tissue restitution: some qualifications and doubts. Clinical Science, 65, 569-578.

Horne, J.A. (1985). The functional significance of human slow wave sleep (h SWS). In Koella, W. P. et al. (Eds) Sleep 1984, Fischer, Berlin.

Horne, J.A., and Moore, V.J. (1985). Sleep EEG effects of exercise with and without additional body cooling. Electroencephalography and Clinical Neurophysiology, 60, 33-38.

Horne, J.A. and Porter, J.M. (1975). Exercise and human sleep. Nature, 256, 573 -575.

Horne, J.A. and Reid, A.J., (1985). Night-time sleep EEG changes following body heating in a warm bath. Electroencephalography and Clinical Neurophysiology, 60, 154-157.

Horne, J.A. and Shackel, B.S. (1985). Heating induced SWS rise - decays with interim wakefulness, inhibited by aspirin. Sleep Research Society, Seattle Meeting 1985.

Horne, J.A. and Staffe, L.H.E. (1983). Exercise and sleep: Body heating effects. Sleep, 6, 36-46.

Johnson, L.C. (1969). Psychological and physiological changes following total sleep deprivation. In Kales A. (Ed) Sleep: Physiology and Pathology. Philadelphia, Lippincott, 206-220.

Kales, A., Heuser, G., Jacobson, A., Hales, J.D., Hanley, J., Zweizig, J.R. and Paulson, M.J. (1967). All night sleep studies in

hypothyroid patients before and after treatment. Journal of Clinical Endocrinology, 27, 1593-1599.

Karacan, I., Williams, R.L., Finley, W.W. and Hursch, C.J. (1970). The effects of naps on nocturnal sleep: Influence on the need for stage 1 REM and stage 4 sleep. Biological Psychiatry, 2, 391-399.

Karacan, I., Rosenbloom, A.L., Londono, J.H., Salis, P.J., Thornby, J.I. and Williams, R.I. (1973). The effect of acute fasting on sleep and the sleep-growth hormone response. Psychosomatics, 14, 33-37.

Keppel, G. (1982). Design and analysis: A researchers handbook (2nd Ed). New Jersey. Prentice Hall.

Kleitman, N. (1963). Sleep and Wakefulness (2nd Ed) Chicago, Chicago University Press.

Kreider, M.B., Busbirk, E.R. and Bass, D.E. (1958). Oxygen consumption and body temperature changes during the night. Journal of Applied Physiology, 14, 765-767.

Lacey, J.H., Crisp, A.H., Kaluccy, R.S., Hartmann, M.K. and Chen, C.N. (1975). Weight gain and sleeping EEG. British Medical Journal, 4, 556-558.

Lamb, D.R.(1984). Physiology of Exercise (2nd.Ed.) New York: McMillan Pub. Co.

Lemon, P.W. and Nagle, F.J. (1981) Effects of exercise on protein and amino acid metabolism. Medicine and Science in Sports and Exercise, 13, 141-149.

Lobstein, D.D., Ismail, A.H., and El-Naygar A.M. (1982). Circulating lipoprotein-cholesterol and multivariate adaptation to regular exercise training of middle-aged men. Journal of Sports Medicine, 22, 440-449.

McFaydyen, U.M., Oswald, I. and Lewis, S.A. (1973). Starvation and human slow-wave sleep. Journal of Applied Physiology, 35, 391-394.

McGinty, D.J. and Drucker-Colin, R.R. (1982). Sleep mechanisms: Biology a control of REM sleep. International Review of Neurobiology, 23, 391-436

Maloletnev, V.I., Telia, Z. A. and Tchatchanashvili, M.G. (1977). Changes in the structure of night sleep in man after intensive muscular work. Sechenov Physiological Journal of the USSR, 63, 11-20.

Maron, M.B. and Horvath, S.M. (1978) The marathon: A history and review of the literature. Medicine and Science in Sports and Exercise, 10, 137-150.

Montgomery, I., Trinder, J., Paxton, S.J. (1982). Energy expenditure and total sleep time: Effect of physical exercise. Sleep, 5, 159-168.

Montoye, H,J., Metzner, H.L., Keller, J.B., Johnson, B.C. and Epstein, F.H. (1972). Habitual physical activity and blood pressure. Medicine and Science in Sports, 4, 175-181.

Moses, J., Lubin, A., Naitoh, P. and Johnson L. C. (1977). Exercise and sleep loss: Effects on recovery sleep. Psychophysiology, 14, 414-416.

Oswald, I. (1962). Sleeping and Waking. New York: Elsevier.

Oswald, I. (1969) Human brain protein, drugs and dreams. Nature, 223, 893-897.

Oswald, I. (1974). Sleep. Penguin Books.

Oswald, I. (1976). The function of sleep. Postgraduate Medical Journal, 52, 15-18.

Oswald, I. (1980). Sleep as a restorative process: Human clues. Progress in Brain Research, 53, 279-288.

Parker, D.C., Rossman, L.G. and Vanderlaan, E.F. (1972). Persistence of human growth hormone release during sleep in fasted and non isocalorically fed normal subjects. Metabolism, 21, 241-252.

Parker, D.C., Sassin, J.F., Mace. J.W., Gotlin, R.W. and Rossman, L.G. (1969). Human growth hormone release during sleep: Electroencephalographic correlation. Journal of Clinical Endocrinology, 29, 871-874.

Parmeggiani, P.L. (1977). Interaction between sleep and thermoregulation. Waking and Sleeping, 1, 123-132.

Parmeggiani, P.L. (1980). Temperature regulation during sleep: A study in homeostasis. In: Orem, J., Barnes, C.D. (Eds) Physiology in Sleep. New York : Academic Press.

Passmore, R., and Durnin, J.V. (1955). Human energy expenditure Physiological Review, 35, 801-840.

Paxton, S.J., Montgomery, I., Trinder, J., Newman, J., and Bowling, A. (1982). Sleep after Exercise of Variable Intensity in Fit and Unfit Subjects. Australian Journal of Psychology, 34, 289-296.

Paxton, S.J., Trinder, J. and Montgomery, I. (1983). Does aerobic fitness affect sleep. Psychophysiology, 20, 320-324.

Paxton, S.J., Trinder,J., Montgomery, I.M., Oswald, I., Adam, K., and Shapiro, C. (1984a). Body Composition and Human Sleep. Australian Journal of Psychology, 36, 181-189

Paxton, S.J., Trinder, J., Shapiro, C.M., Adam, K., Oswald, I. and Graf, K.J. (1984b). Effects of physical fitness and body composition on sleep and sleep related hormone concentrations. Sleep, 7, 339-346.

Phillips, F., Chen, C.N., Crisp, A.H., Koval, J., McGuinness, B., Kaluccy, R. S., Kaluccy, E.C., and Lacey, J. H. (1975). Isocaloric diet changes and electroencephe during sleep in fasted and non isocalorically fed normal subjects. Metabolism, 21, 241-252.

Rechtschaffen, A and Kales, A. (Eds). (1968). A Manual of Standardized Terminology, Techniques and Scoring system for Sleep Stages of Human Subjects. Washington D.C. U.S. Government Printing Office.

Roffwarg, H., Muzio, ,J.N., and Dement, W.C. (1966). Ontogenetic development of human sleep-dream cycle. Science, 152, 604-619.

Samet, J.M. and Chick, T.W. (1983). Exercise and the Lung. In Appenzeller, O., and Atkinson, R. Sports Medicine: Fitness, Training, Injuries. 2nd. Ed. Baltimore-Munich: Urban & Schwarzenberg. 211-227.

Sassin, J.F., Parker, D.C., Mace, J.W., Gotlin, R.W., Johnson, L.C. and Rossman, L.(1969). Human growth hormone release: relation to slow-wave sleep and sleep-waking cycles. Science, 165, 513-515.

Schade, D.S. (1983). Stress Hormone Response to Exercise. In Appenzeller O. and Atkinson, R. Sports Medicine: Fitness, Training, Injuries. 2nd. Ed. Baltimore-Munich: Urban & Schwarzenberg,149-156.

Shapiro, C.M. (1982). Energy expenditure and restorative sleep. Biological Psychology, 15, 229-239.

Shapiro, C.M., Griesel, R.D., Bartel, P,R,. & Jooste, P.L. Sleep patterns after graded exercise. Journal of Applied Physiology, 1975, 39, 187-190.

Shapiro, C.M. Bortz, R.D., Bartel, P.R. and Jooste, P.L. (1981). Slow-wave sleep: A recovery period after exercise. Science, 214, 1253-1254.

Simonson, E. (1971). Physiology of Work Capacity and Fatigue. Springfield, Illinois: Charles C. Thomas.

Snyder, F. (1966). Toward an evolutionary theory of dreaming. The American Journal of Psychiatry, 123, 121-142.

Spiegel, R. (1981). Sleep and Sleeplessness in Advanced Age. MTP Press limited, Lancaster, England.

Takahashi,Y., Kipnis, D.M. and Daughaday, W.H. (1968), Growth hormone secretion during sleep. Journal of Clinical Investigations, 47, 2079-2090.

Thomas, V. (1975). Exercise Physiology. London: Crosby Lockwood Staples. Torsvall, L. (1981). Sleep after exercise: A literature review. Journal of Sports Medicine and Physical Fitness, 21, 218 - 225.

Torsvall, L. Akerstadt, and Lindbeck, J. (1984). The effects on sleep stages and EEG power density of different degrees of exercise in fit subjects. Electroencephalography and Clinical Neurophysiology, 57, 347-363.

Trinder,J., Bruck, D., Paxton, S.J., Montgomery, I. and Bowling, A. (1982). Physical Fitness, exercise, age and human sleep. Australian Journal of Psychology, 34, 131-138.

Trinder, J., Paxton, S.J., Montgomery, I., and Fraser,G. (1985). Endurance as opposed to power training: their effect on sleep. Psychophysiology, 22, 668-673.

Verdone, P. (1968). Sleep satiation: Extended sleep in normal subjects. Electroencephalography and Clinical Neurophysiology, 24, 417-423.

Walker, J.M. and Berger, R.J. (1980). Sleep as an adaptation for energy consumption functionally related to hibernation and shallow topor. Progress in Brain Research, 53, 255-278.

Walker, J.M., Floyd, T,C., Fein, G., Cavness, C., Lualhati, R., and Feinberg, I. (1978). Effects of exercise on sleep. Journal of Applied Physiology, 44, 945-951.

Webb, P. and Hierstand, M. (1975). Sleep, Metabolism and Age. Journal of Applied Physiology, 38, 257-262.

Weitzman, E.D., Zimmerman, J.C., Czeisler, C.A. and Ronda, J. (1981). Cortisol is inhibited during sleep in normal man. Sleep Research, 10, 107.

Wells, D.T., Allan, R.P. and Wagman, A.M.I. (1977). A single-channel system of recording eye movements. Psychophysiology, 14, 73-74.

Williams, R.L., Karacan, I., and Hursch, C. (1974). EEG of human sleep: clinical applications. John Wiley and Sons, New York.

Wright, T.W., Zauner, C. W. and Cade, R. (1982). Cardiac output in male middle aged runners. Journal of Sports Medicine, 22, 17-22.

Zepelin, H. (1983). Normal age related change in sleep. Advances in Sleep Research, 8, 431-444.

Zepelin, H. and Rechtschaffen, A (1974). Mammalian sleep, longevity and energy metabolism. Brain, Behaviour and Evolution, 10, 425-470.

Zir, L.M., Smith, R.A. and Parker, D. (1971). Human growth Hormone release in sleep: Effect of daytime exercise. Journal of Clinical Endocrinology, 32, 662-665.

APPENDIX 1

EVENING QUESTIONNAIRE

MORNING QUESTIONNAIRE

EVENING SLEEP QUESTIONNAIRE

NAME: _____

DATE: _____

GROUP: _____

CONDITION: _____

For each of the questions below put a mark across the line at that position which represents how you feel.

To what extent do you feel physically tired?

Not at all _____ a great deal

To what extent do you feel mentally tired?

Not at all _____ a great deal

To what extent do you feel physically stressed?

Not at all _____ a great deal

To what extent do you feel mentally stressed?

Not at all _____ a great deal

MORNING SLEEP QUESTIONNAIRE

NAME: _____

DATE: _____

GROUP: _____

CONDITION: _____

For each of the questions below put a mark across the line at that position which represents how you feel.

Have you had a good night's sleep?

Good night's sleep _____ Bad night's sleep

Have you had a restful night's sleep?

Restful sleep _____ Disturbed sleep

Have you slept deeply?

Slept Lightly _____ Slept Deeply

Do you feel refreshed from your night's sleep?

Feel refreshed _____ Feel fatigued

APPENDIX 2

PUBLICATIONS

Paxton, S. J., Montgomery, I., Trinder, J., Newman, J., Bowling, A., 1982. Sleep after exercise of variable intensity in fit and unfit subjects, Australian journal of psychology, 34(3), 289–296
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Montgomery, I., Trinder, J., Paxton, S., Fraser, G., 1985. Sleep disruption following a marathon, Journal of sports medicine and physical fitness, 25(1/2), 69/74

APPENDIX 3

TABLE 1

Mean duration and standard deviation in minutes of selected sleep variables for fit subjects as a function of age and exercise.
Table 7.2 expanded

TABLE 2

Mean duration and standard deviations in minutes for non exercise nights as a function of age and level of fitness.
Table 7.3 expanded

Table 1

Mean duration and standard deviation in minutes of selected sleep variables for fit subjects as a function of age and exercise. The data for each of the two consecutive nights in each condition has been given separately.

Variable	Younger (N=10)				Older (N=12)				Significant Effect
	No Exercise		Exercise		No Exercise		Exercise		
	1	2	1	2	1	2	1	2	
TIB	596.8 (43.2)	614.9 (49.7)	620.0 (50.2)	612.2 (54.9)	583.0 (96.3)	556.4 (84.0)	574.8 (69.5)	579.6 (69.6)	AEN*
TST	542.4 (58.6)	557.9 (58.9)	566.2 (73.7)	535.4 (57.1)	527.7 (86.8)	502.4 (70.8)	532.3 (66.6)	496.0 (50.9)	N*
SPT	566.1 (58.9)	584.4 (52.6)	597.3 (57.6)	631.4 (70.6)	559.9 (89.6)	522.4 (78.6)	549.5 (69.2)	542.6 59.8)	
SOL	12.1 (5.4)	21.7 (9.4)	16.4 (12.4)	28.5 (21.4)	12.7 (8.0)	14.6 (9.0)	15.1 (9.6)	28.7 (28.9)	E*,N*
TTA in TIB	54.4 (31.0)	57.1 (41.4)	53.8 (45.4)	76.8 (68.2)	55.3 (41.1)	54.0 (32.0)	42.5 (17.5)	83.5 (44.6)	N*, EN*
W+MT+1 in TIB	113.0 (33.8)	126.2 (47.7)	122.2 (49.0)	148.9 (68.0)	122.0 (52.0)	124.8 (52.0)	114.3 (29.4)	165.8 (51.7)	E*,N**,EN*
Sleep Efficiency	.909 (.056)	.907 (.068)	.913 (.081)	.875 (.095)	.905 (.065)	.903 (.053)	.926 (.030)	.856 (.067)	N*
Stage 2	276.5 (32.5)	280.3 (53.8)	286.0 (59.4)	287.7 (45.9)	296.1 (72.3)	277.3 (53.7)	301.9 (61.9)	276.9 (52.6)	

A = Age; E = Exercise; N = Nights; * = significant < .05; ** = significant <.01; Sleep variables are defined in the text.

Table 1 (continued)

Mean duration and standard deviation in minutes of selected sleep variables for fit subjects as a function of age and exercise. The data for each of the two consecutive nights in each condition has been given separately.

Variable	Younger (N=10)				Older (N=12)				Significant Effect
	No Exercise		Exercise		No Exercise		Exercise		
	1	2	1	2	1	2	1	2	
Stage 3	31.0	37.4	39.3	32.4	29.4	26.9	32.1	28.8	
	(14.8)	(13.7)	(18.7)	(8.7)	(11.8)	(7.5)	(8.7)	(11.5)	
Stage 4	57.9	48.8	52.9	38.4	14.5	12.6	15.8	11.7	A*,E*,N**,AE*
	(13.1)	(14.1)	(13.3)	(19.4)	(21.8)	(17.1)	(21.6)	(15.2)	
Stage 3+4	88.9	86.2	92.2	70.8	43.9	39.5	47.9	40.5	A*,*N**,AE*,EN*
	(21.7)	(18.8)	(27.8)	(24.8)	(26.6)	(22.4)	(26.0)	(21.6)	
3+4 in 1st 150 mins	61.2	56.2	55.9	48.2	31.8	27.8	30.3	25.3	A**,E*.N*
	(15.3)	(8.0)	(15.0)	(15.9)	(17.1)	(18.2)	(11.3)	(14.6)	
Stage 2+3+4	365.4	366.5	378.2	358.5	340.0	316.8	349.8	317.4	N**
	(36.5)	(47.0)	(47.7)	(43.8)	(56.8)	(44.2)	(47.3)	(36.1)	
REM	119.1	123.0	118.6	105.3	121.0	114.8	110.7	96.2	E*
	(26.4)	(18.8)	(36.3)	(19.2)	(29.0)	(24.5)	(32.0)	(15.2)	
REM in 1st 150 mins	8.4	13.5	9.2	9.0	14.9	14.5	10.5	11.8	E*
	(5.5)	(4.6)	(6.4)	(8.0)	(8.4)	(8.8)	(6.5)	(9.5)	

A = Age; E = Exercise; N = Nights; * = significant < .05; ** = significant <.01; Sleep variables are defined in the text.

Table 2

Mean duration and standard deviation in minutes of selected sleep variables for non exercise nights as a function of age and level of fitness. The data for each of the two consecutive nights in each condition has been given separately.

	Younger				Older				
Variable	Fit (N=10)		Unfit (N=10)		Fit (N=12)		Unfit (9)		Significant Effect
	1	2	1	2	1	2	1	2	
TIB	596.8	614.9	592.1	554.1	583.0	556.4	544.0	523.8	
	(43.2)	(49.8)	(51.3)	(62.2)	(96.4)	(84.0)	(50.5)	(59.1)	
TST	542.4	557.9	544.7	507.4	527.7	502.4	497.9	469.9	A*N*
	(58.6)	(58.9)	(57.2)	(51.1)	(86.8)	(70.8)	(68.2)	(60.0)	
SPT	566.1	584.4	562.8	521.1	559.9	522.4	517.8	495.9	N*
	(58.9)	(52.6)	58.3)	(59.7)	(89.6)	(78.6)	(67.6)	(71.2)	
SOL	12.1	21.7	28.7	30.7	12.7	14.6	21.7	17.9	F*
	(5.4)	(9.4)	(29.2)	(14.8)	(8.0)	(9.0)	(11.8)	(9.7)	
TTA in TIB	54.4	57.1	47.5	46.7	55.3	54.0	46.1	53.8	
	(31.0)	(41.4)	(35.5)	(28.5)	(41.1)	(40.0)	(28.7)	(24.2)	
W+MT+1 in TIB	113.0	126.2	111.1	105.8	122.0	124.8	117.4	138.6	AFN*
	(33.8)	(47.7)	(39.2)	(33.4)	(52.0)	(52.0)	(30.5)	(25.9)	
Sleep Efficiency	.909	.907	.920	.916	.905	.903	.915	.897	
	(.056)	(.068)	(.058)	(.042)	(.065)	(.053)	(.060)	(.046)	
Stage 2	276.5	280.3	277.7	248.4	296.1	277.3	296.8	274.2	N*
	(32.5)	(53.8)	(27.8)	(51.3)	(71.3)	(53.7)	(62.4)	(62.0)	

A = Age; F = Fitness; N = Nights; * = significant < .05; ** = significant <.01; Sleep variables are defined in the text.

Table 2 (continued)

Mean duration and standard deviation in minutes of selected sleep variables for non exercise nights as a function of age and level of fitness. The data for each of the two consecutive nights in each condition has been given separately.

Variable	Younger				Older				Significant Effect
	Fit (N=10)		Unfit (N=10)		Fit (N=12)		Unfit (9)		
	1	2	1	2	1	2	1	2	
Stage 3	31.0 (14.7)	37.4 (13.7)	31.0 (12.5)	29.4 (6.6)	29.4 (11.8)	26.9 (7.5)	16.8 (15.2)	18.2 (17.5)	A*F*
Stage 4	57.9 (13.1)	48.8 (14.1)	46.2 (13.3)	46.0 (19.4)	14.5 (21.8)	12.6 (17.1)	6.1 (11.3)	3.3 (4.7)	A*
Stage 3+4	88.9 (21.7)	86.2 (18.8)	77.2 (27.7)	75.4 (23.8)	43.9 (26.6)	39.5 (22.4)	22.9 (25.6)	21.5 (21.6)	A**,F*
3+4 in 1st 150 mins	61.2 (15.3)	56.2 (8.0)	49.4 (19.7)	52.2 (15.0)	31.8 (17.1)	27.8 (18.2)	17.9 (23.5)	17.1 (17.8)	A**,F*
Stage 2+3+4	365.4 (36.5)	366.5 (47.0)	354.9 (25.2)	323.8 (41.3)	340.0 (56.8)	316.8 (44.2)	319.7 (53.2)	295.7 52.9)	A*,N*
REM	119.1 (26.4)	123.0 (18.8)	127.5 (34.1)	125.4 (13.6)	121.0 (29.0)	114.8 (24.5)	106.8 (22.7)	86.7 (25.4)	A*
REM in 1st 150 mins	8.4 (5.5)	13.5 (4.6)	13.5 (14.2)	13.3 (9.3)	14.9 (8.4)	14.5 (8.8)	22.1 (9.4)	13.8 (9.8)	A*

A = Age; F = Fitness; N = Nights; * = significant < .05; ** = significant <.01; Sleep variables are defined in the text.